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ABSTRACT

Noting that linguistics and the neurological sciences have developed independently, this paper presents a coordinated approach to man's understanding of language, cognition, and mind. A neurological model is developed following a discussion of the rationale of such an approach. Chapters include: (1) the relation of neurological evidence to models of language, (2) a schematic model of language, (3) functional anatomy of the central language system, (4) functional anatomy of the peripheral language modalities, and (5) concluding remarks. Tables and charts illustrate theory. Bibliographical references are included. (RL)

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A MODEL FOR NEUROLINGUISTICS

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1. Introduction

The written word and the acoustic waveform of human language are representatives, albeit imperfect, of neurolinguistic units in the peripheral and central nervous systems. It is generally recognized that in themselves they cannot provide sufficient information to account for man's knowledge and use of language. Consequently, the construction of a model of language will perforce rely not only upon abstractions (rules) that can be consistently related to graphic/acoustic data, but also upon constraints that are derived in part from other disciplines. Language is in no more of a neurological vacuum than it is in a psychological or cultural vacuum; consequently the best model of language will incorporate as broad a range of interdisciplinary facts as can be consistently related to each other. Linguistics, as it is understood here, is the science of investigating the mechanisms which underlie or are responsible for the observed language behavior of human beings. A linguistic hypothesis thus is a statement about one or more of these mechanisms. Neurolinguistics differs from linguistics *per se* by implying a broadened empirical domain that considers, as evidence for linguistic hypotheses, various observables not usually entertained by the linguist - in particular, aphasic speech, and in general, the neuroanatomical substrates of the aphasias (see Green [1969, 1970] for further discussion). Neurolinguistics will constrain the account of the mechanisms underlying language behavior to those which may correspond to neurological systems, hypotheses and data in the broadest sense - i.e., neuroanatomy, neurophysiology, neurochemistry, neuropsychology (in short, the neurological sciences) can and ought to make appropriate contributions to our understanding of linguistic structure.

It is quite true that linguistics and the neurologic sciences have developed quite independently of each other to date and there is no reason to assume that they cannot continue to do so. Nevertheless most serious researchers in the aforementioned disciplines would agree that the ultimate understanding of language, congnition and mind will absolutely depend upon the coordination of all avenu of approach. Even though this assertion is easily accepted, it could be useful to delineate some of the reasons why it is true. Linguists as well as others have often claimed that there is a significant qualitative difference between human language and animal communicatio systems. A review of some of these claims can be found i Drewe, Ettlinger, Milner and Passingham [1970]. Some elegant and quite convincing hypotheses have been marshalled to substantiate these claims. Recently howeve some psycholinguistic experimentation with chimpanzees, e.g. D. Premack (University of California at Santa Barba and R.A. and B.T. Gardner (University of Nevada), has raised some important new questions about the capacity of infra-human primates to acquire language. No matter how the psycholinguistic arguments are resolved, nor for that matter how the vocal-tract morphological arguments are resolved (cf. Lenneberg [1967]), the ultimate decision concerning that capacity will have to be made on the basi of brain structure and function. Language could not be structured in such a way as to be incompatible or un-acceptable to the brain; otherwise, it would be impossibl to learn it, know it or use it, much less think about it. But of course we want to know even more than this, namely what aspects of linguistic structure are innate, species-specific and 'biological' and what aspects are acquired, common to all communication systems and 'social'. It is absurd to suppose that these questions can be answered within a purely linguistic framework and of course it cou hardly be said that an understanding of language may be reached without the answers.

In addition to such obvious justifications as above, there may be more practical and immediate justifications for undertaking a neurolinguistic model. The analytical power of current linguistic hypotheses well exceeds the known facts of language and consequently it is important for the construction of an adequate grammar that it be subject to constraints on the domain, applicability and type of rules of which it is comprised. This was observe several years ago by Chomsky [1967] who remarked that

> ... sooner or later ... it is going to be necessary to discover conditions on theory construction, coming presumably from experimental psychology or from neurology, which will resolve the alternatives that can be arrived at by the kind of speculative theory construction linguists can do on the basis of the data available to them. (100)

Finally, consider this goal from the perspective of the neurological sciences: one of the primary purposes for investigating physiological and neurological mechanisms and their associated anatomical substrates is to understand the basis of human (and animal) behavior. However, to put it in a misleadingly simple way, behavior is no more differentiated than the electrical activity of the central nervous system. When the neurological sciences arrive at some understanding of the processes that underlie the physical data obtained from the nervous system, it will be imperative that a science of behavior has developed far enough to explain what such processes mean. The question might be: will it make sense at that point to explain a specific configuration of neuronal spike trains as a representative or code for a specific word? Bearing in mind the fact that any such explanation is oversimplified, it is nevertheless the case that we must assume there to be a correlation between behavioral phenomena external to the organism and neurological phenomena internal to the organism. Discovering what this relationship might be is the task of all the sciences of man.

2. The Relation of Neurological Evidence to Models of Language

There are two preliminary questions of some importanc which need to be answered: (1) given an agreement on an ultimate goal to correlate linguistics and neurological sciences, how in fact can hypotheses and data from several different models and paradigms be correlated? and (2) have we currently available a sufficient body of hypotheses and data that, presuming a favorable answer to the first question, a model can be proposed within which such correlations might be made? The answer to the first question largely depends upon what particular position one wishes to take with respect to the philosophy of science; a mild form of materialism, subject to certain qualifications, is the view taken in this paper. The answer to the second question is, because of the nature of the materialist position espoused, a qualified yes; the major part of this paper will be devoted to an elaboration of a neuroanatomical model for neurolinguistics. A great deal of material relevant to this task will be either assumed or ignored; it is not possible to review the literature on aphasiology, functional neuroanatomy, physiological psychology, linguistics or neurology. Because the perspectives being presented here are an attempt to synthesize divergent materials, the references both in the text and in the bibliography have been selected to extend pertinent discussions rather than document each proposal. More complete references may be found in Whitaker [1969, 1970, to appear].

Fodor [1968] discusses two types of materialism that might be embraced by the contemporary psychologist. The first is the stronger type - reductionism - whereby the facts of the neurological sciences are taken as a micro-analysis of the hypotheses of psycholinguistics. In such a view, one would establish some principle of verbal behavior, e.g. that the relative strength of word associations depends upon the proximity of the words, and assume that this is re-statable in terms of neuronal spike trains or neurochemical processes. Fodor rejects the straight reductionist position; his rejection is well-motivated. The second and milder version of materialism is the proposal that it is possible to establish functional equivalences between disciplines. This position, the one adopted in this paper, holds that psycholinguistic mechanisms/hypotheses may be directly correlated with *functionally equivalent* (i.e. in the model) mechanisms/hypotheses in the neurological sciences. To the extent that such correlations can be plausibly made, the hypotheses and mechanisms in both disciplines are strongly supported; to the same extent, the general neurolinguistic model of language is supported. But in addition one might expect that weak or poorly-substantiated hypotheses in any one discipline may gain in credibility to the extent that they correlate with their functional equivalents in other disciplines or the general model as a whole. Unfortunately, it is a bit too easy to overstate this last point: most of the researchers in all the disciplines mentioned above have been more than careful to stress the general lack of knowledge (lack of suitable, integrated hypotheses) about language, cognition and mind. This is not disputed - we do know very little. As a general caveat for what follows, it might be well to observe that virtually the only thing that is certain is that language is represented in the human brain - the hypotheses, mechanisms and even the facts from linguistics and the neurological sciences are only the best proposals that can be made by the author at the present time.

Accepting the position of mild materialism just advanced forces one to consider some contingent issues: (1) localization *vs.* non-localization of cortical functions, (2) rules *vs.* strategies and (3) competence/performance and the level of abstraction. The debate between

localization and non-localization of cortical functions has been seriously contested for over a century. It is obvious that to accept the notion of functional equivalences for an *anatomical* model for neurolinguistics is to accept some form of localization of cortical function. The arguments which are most pertinent can be reviewed briefly. A non-localizationist position maintains a unitary loss, or single element deficit, view of aphasia (see Schuell, Jenkins and Jimenez-Pabon [1964] or Lenneberg [1967] for discussion); in the author's view neither the clinical, the psychological, nor the linguistic analysis of aphasia supports this (see Green [1969b, 1970], Geschwind [1965], Nielsen [1946], Hécaen [1969], Luria [1966], Riklan, Levita, Zimmerman and Cooper [1969], Penfield and Roberts [1959], Newcombe [1969], Whitaker [1968, 1969a, 1969b] for discussion).

A non-localizationalist position often minimizes both the anatomical differences between cortical areas (e.g. preponderance of pyramidal cells in motor cortex and and granular cells in sensory cortex) and the anatomical connections within each hemisphere (cortico-cortical, thalamo-cortical, etc.) whereas the localizationist position stresses these distinctions (see Luria [1966], Geschwind [1964, 1965], Riklan and Levita [1969] and Penfield and Roberts [1959] for further discussion ; since the localizationist position, with qualifications, is accepted in this paper, further discussion of neuroanatomy will be taken up later).

The non-localizationist position can point to the fact that in the literature on aphasia there are many inconsistencies in the putative loci of lesions associated with specific defects (see Lenneberg [1967] for discussion) Although this is factually true, the reasons for the apparent refutation of the localizationist position are easy to pinpoint and once this is done most, although not all, of the inconsistencies disappear. For example, Penfiel and Roberts did electrical stimulation 'mapping' on brains that not only were diseased but had been diseased for a long time prior to surgery; some functional reorganization and compensation would have taken place. In addition, it is unclear how large the electrical current was that was introduced via the electrode and it is unclear whether any attempt was made to discount the effects of after-discharges. Despite all these shortcomings, the evidence obtained by Penfield and Roberts is remarkable for its support of localizationist views. In other studies lesions can and have been located by gross inspection of the surface of the cortex, gross inspection of serial sections of the brain, microscopic inspection with or without

staining techniques and, in some instances, unfortunately, without even looking directly at the brain. Very few attempts to locate lesions (in man) have made use of cytoarchitectural information (as, for example, postulated by Brodmann on the basis of cell densities and cell-type layers in the cortical mantle) but have instead relied upon topographical information (the gyri and sulci). This alone would introduce many problems in localization since the gyri and sulci are nearly as variable as fingerprints or voiceprints from person to person. Even so, localizations based on brain topography are remarkably consistent. One might well ask how it is that lesion loci so often correlate with deficits than the more usual question of why there fails to be a perfect match. In this paper a strict localizationist position is rejected: it is not claimed that all cortical functions have a single, discrete locus. What is claimed is that certain cortical (and sub-cortical) areas of the brain can be functionally equated with certain psycholinguistic hypotheses and mechanisms. These cortical areas are not perfectly demarcated partly because the distribution of cytoarchitectural distinctions itself is not perfectly demarcated; furthermore, there is the possibility of limited functional re-organization of the cortex that may further blur the boundaries of functional areas based on lesion evidence. And last, it is not likely to be the case that a single cortical locus of any description (cell, network or 'lobe') will be the single substrate or correlate for a discrete linguistic unit or rule. If words are stored in the brain it is probable that they are 'content-addressed' rather than 'location-addressed'. With the above qualifications, the localizationist view of brain functions is accepted.

Investigation in linguistics has led to the formalization of hypotheses as rules (and rule-sets) which operate on linguistic units in specified ways. The search for psychological support for these rules has met with limited success (see Watt [1970] for a review); there is more encouraging support for the units of the grammar. It is reasonable to ask whether or not the brain's usual mode of functioning is as fully categorial as would be required by a rule-governed model, or whether brain functions are (in Azcoaga's [1970] terms) 'dynamic processes' (see also Luria [1966]).

A rule-governed operation is one in which all features of a given signal are computed in full. For a perceptual operation such a model requires the complete analysis of the signal and a one-one correspondence between the full analysis and the storage signal being matched to the message signal. A strategy-governed operation is one in

which the features of a given signal are hierarchically arranged in order of communicative importance (the features *may* be so arranged in a rule-governed operation, too, but such an arrangement would be irrelevant for perceptual analysis). For a strategy-governed perceptual operation the model requires only that a sufficient number of the features of the signal match with corresponding features of the storage signal to allow a probable guess. Since the context can often specify in advance some of the major features of the signal, the strategies may be employed in the usual case for merely analyzing the minor 'distinguisher' features. The two models have analogous distinctions for motor production operations. It should be clear that, given a more predictable signal due to an appropriate context, the strategy-governed model is much faster. Without an appropriate context the strategy model may require as long or longer time for operation than its corresponding rule-governed model. Although there is at present little evidence which may throw light on this issue, a plausible model may be developed either way without violating the requirements of functional equivalence stated earlier. Specifically, it is possible that the brain requires a complete deep structural representation of a sentence in order to encode or decode it, in which case the functional equivalences would be rather direct. It is more likely that the brain computes, on the hierarchical principle, just enough information about the deep structure representation of the sentence that would facilitate a well-motivated (statistically highly probable) guess. This view would place more importance on contextual information beyond the level of the sentence than in the rule-governed model, since such information would be required in order to maximize the success of the strategies. In either view of brain function, the equivalence is established with formal, discrete linguistic rule; if the correspondence fails because the neurologically more adequate model is strategy-governed, then it is necessary to incorporate the larger information domain as well as probability principles at that level in the general model. At present this issue is of more significance in computer simulation research than in seeking some fundamentals of an anatomically-oriented neurolinguistic model.

The distinction between competence and performance appears to have been quite useful in linguistic theory (Chomsky [1965]), less useful in psycholinguistic theory (Lyons and Wales [1966]), somewhat confusing in neuro-linguistic theory (Weigl and Bierwisch [1970], Traill [1970]) and virtually irrelevant to phonetic theory. Linguists might maintain that neurolinguistics is in fact a thinly disguised theory of performance; to do so,

however, produces a theory of competence so highly abstract that it is difficult to determine how it might relate to psychological and neurological hypotheses. If the notion of functional equivalence is to have any meaning, some attempt must be made to find levels in the respective models which can be correlated. As Reich [1968] observed, it can be uncomfortable when parameters of verbal behavior are found under plausible experimental conditions and those parameters appear to contradict the linguistic model (see also Watt [1970] in this regard). It is not acceptable to remove linguistic hypotheses to an abstract level outside the domain of neurological correlation and refutation. This does not remove a degree of responsibility from the neurological sciences: the model must be adequate from both ends and a failure to match a linguistic hypothesis may not be due to its abstractness but to shortcomings in the neurological model. At the same time, the general neurolinguistic model should provide for a separation of the problem-solving ability, the ability to assume the 'abstract attitude', general cognitive abilities (including visuo-spatial perception) and the linguistic ability. The motivation for making such distinctions is two fold: first, the neurolinguistic model should not account for abilities other than language even though it should be able to integrate such abilities in the more comprehensive theory of mind, and second, it is likely that problem-solving and general cognitive abilities (intelligence) obey Lashley's mass-action and equipotentiality parameters of brain function, i.e. are non-localized. In quite parallel fashion, one would not require a neurolinguistic model to provide an account of emotional mechanisms or general visual perception (although the latter system will play a role in the immediate context of the model as will be explained below), even though these systems are if anything more anatomically localized than the language system.

An issue which is not contingent upon accepting the position of mild materialism but which is of equal importance to the overall philosophical assumptions here, is the matter of basing hypotheses of normal function upon the data obtained from abnormally functioning organisms - the 'lesion' assumption. Lenneberg [1967] suggests that lesion evidence is suspect because of the variety of lesion types, some of which may affect larger areas of the brain than is generally conceded, because some lesions may not in fact totally disrupt the affected area but introduce unknown behavioral concomitants, because the exact locus of some lesions is difficult to ascertain, and because (as noted above) lesion studies in the literature do not completely correspond. First, it must be recognized that

brain lesions of any type are not always exactly circumscribed in neural tissue, are not always easily identified and are not always strictly and completely correlated with functional deficits - all neurologists recognize this. Nevertheless the statistical correlations accumulated over the past century are sufficiently impressive that a plausible case can be made (and in fact is made by most neurologists, neurosurgeons and speech pathologists) for lesion evidence in support of the localizationist position, as qualified above. The objections to lesion evidence may be met with the following brief and necessarily incomplete summary.

The usual lesions studies are the stroke (CVA) and the traumatic injury due to penetrating missiles because of their higher frequency of occurrence. The CVA may incompletely destroy the affected tissue, resulting in some functional recovery; a plateau is reached eventually after which time permanent functional losses can be assessed. Vascular lesions in general are ultimately good sources of localization since the infarcted area is clearly recognizable on histological examination. Traumat injury temporarily disrupts wider areas of the brain but usually quickly stabilizes; infection, of course, will change this pattern. It is often the case that the residu defect from a missile wound is of a highly specific but no seriously debilitating nature; such deficits require sophisticated testing procedures to detect. An excellent review of the literature and a contemporary analysis which largely supports the position assumed here can be found in Newcombe [1970]. Generalized diseases of the brain such as Parkinsonism are of course not localized; many of them, however, do not result in deficits of the central language system (aphasia) anyway, which in itself is an interesting phenomenon supporting the current model. Tumors are relatively poor sources of data, but this is due more to the fact that they disrupt brain structure at a slow rate thereby allowing for compensation and reorganization, than to the inability to identify histologically the locus of lesion. Aneurysms, hematoma and closed head injuries are difficult to assess behaviorally until after a recovery period and can be difficult to assess structurally; however, few claims for loci and functional correlations are based upon these lesions. Rarer lesion types such as carbon monoxide poisoning can often provide striking support for the model (see Geschwin Quadfasel and Segarra [1968]).

With respect to the observation that many lesions destroy an area too large to be of use in localizationist theories, there is no simple answer. Proposed anatomical

correlations are not generally based upon single cases but rather upon the accrual of a large number of cases. There have been some interesting exceptions to this which in fact later turned out to be strikingly corroborated when more evidence was obtained, e.g. Nielsen's [1946] proposal of a frontal motor center anterior to the pre-central gyrus, above Broca's Area, that is involved in the ability to write. Nielsen discussed a single case, which in fact was not even his own; however, Hécaen [1969] has recently noted four cases of isolated agraphia with lesions in the same general area, providing strong support for Nielsen's claim. To be sure there are a great many unknowns in the clinico-anatomical study of the aphasias; eventually general correlations are demonstrable, correlations which may not fit any single case exactly, but which in the main account for the evidence.

Another argument is often used in criticizing lesion studies: the destruction or extirpation of a particular cortical area which results in a particular behavioral loss or abnormality, may not permit the inference that when the area was normal it had the function which was lost, or worse, the role of suppressing the abnormality. On occasion arguments like this lead to the conclusion that the loss of brain structure can 'add' behavioral functions to the organism, a conclusion which is simply rejected here as being absurd. On other occasions though such arguments reflect on unwillingness to accept the research paradigm - i.e., that one can infer the properties of normal function by examining abnormal function. Since this is an *a priori* assumption, there is little to be said about it other than to invoke the 'authority' of the numerous researchers in the neurological sciences who accept the assumption. There is a further, in fact, serious, criticism of lesion studies which at the moment is un-answerable: it is possible, even likely, that certain aspects of brain function cannot be isolated or disrupted by lesions of the sort investigated so far (and of course certain neurochemical and electrophysiological studies that use animal subjects cannot be replicated using human beings). These non-isolatable functions may be expressible only in terms of electrical or chemical events in the central nervous system which are not subject to gross structural damage in a specific area. That this is a possibility should be a challenge to the neurological sciences to seek other means of obtaining data from the brain, for example, the cortical evoked potential (in this regard, see Donchin and Lindsley [1969]).

Many brain lesions produce loss of or impairment of brain function. Loss or impairment thus provide positive evidence for the neurological reality of the function under

the conditions just noted. This is essentially the basic research paradigm in the neurological sciences: to ablate tissue or otherwise block or inhibit the normal functioning of the tissue by electric shock or chemical reaction. The assumption is that a comparison of pathologic with normal behavior under controlled experimental conditions, reveals function of tissue so manipulated. Virtually the same assumption underlies the neurological investigation of the aphasias upon which much of this study is based. The principal difference is that with the human brain one cannot freely experiment. To consider an accidental lesion which causes aphasia as an experimental datum requires a careful analysis of a wide variety of mitigating circumstances, some of which have already been noted.

A similar situation but with a quite different consequence exists when a brain lesion affects function X when it is function Y that is being investigated. In this case the evidence is negative, for only one of the potentially several functions can be ruled out. For example, if one is looking for evidence that sub-cortical structures such as the basal ganglia or thalamus do or do not play a significant role in the central language system, negative evidence is provided by noting that lesions in these structures produce dysarthria (which is an impairment of muscle innervation) rather than aphasia. There is an additional aspect to this research paradigm which is even more problematic: the distinction between loss and impairment itself. The loss of a particular aspect of brain function might be either a direct loss or loss of access to the storage necessary for the execution of the function. For example, if a certain set of phonological rules no longer characterize the speech output of a brain-damaged patient, it might be that the neurological correlate of these rules was directly destroyed or it might be that a neurological mechanism which activated the correlate in question was destroyed. To rephrase this issue in another framework, it is often difficult to distinguish a loss of a memory store from the loss of access to storage. The nature of *impaired* brain function does shed some light on the problem. A brain-damaged patient's ability to perform tasks often fluctuates from day to day, particularly and primarily in the recovery period following a brain insult. For example, a patient may know a word on one day and not know it on the next, where 'know' can mean he is able to name an object, to match the correct word with its picture, to point to the picture which corresponds to a word, etc. Under such conditions the usual interpretation is that access is impaired not storage. Notice however that under either condition we may infer the neurological reality

of the function in question as well as the fact that the locus of the lesion probably identifies a structure that is involved in some way in the function. Some further remarks on this matter may be found in Weigl and Bierwisch [1970].

3. A Schematic Model of Language

The language system in the brain will eventually be modelled by empirical support from several disciplinary domains, not just simply by our intuitions about linguistic structures. With the difficulties and qualifications cited above well in mind, a box-diagram approach to a general neurolinguistic model may be discussed. In the most simplified perspective, four distinct sub-systems of human language are intuitively clear: speaking and listening, which all human languages have, and reading and writing, which a few human languages have. These systems are distinct because of their individual dependence upon distinct sensory organs - the eye and the ear - and distinct muscular groups - the mouth and the hand. In order to successfully and naturally speak or write, there must be some degree of monitoring (feedback) minimally at a level compatible with the meaning or semantics of the intended production (see Laver [1969]). This high-level feedback naturally links the speaking and listening systems to each other and also links the writing and reading systems to each other; the effects of blocking this feedback are quite obvious on a moment's reflection. But the analogies seem to break down at this point, for, while it is difficult to imagine oneself speaking with any muscle groups other than those of the mouth (vocal tract) or listening with any sense organ other than that of the ear (auditory system), it is *not* difficult to imagine reading with other sensory organs - as with the fingers for braille - or writing with other muscle groups - the non-dominant hand, or with a pen clenched in the teeth, etc. On neuropsychological criteria then we may wish to consider the reading and writing system one step removed in some sense from the speaking and

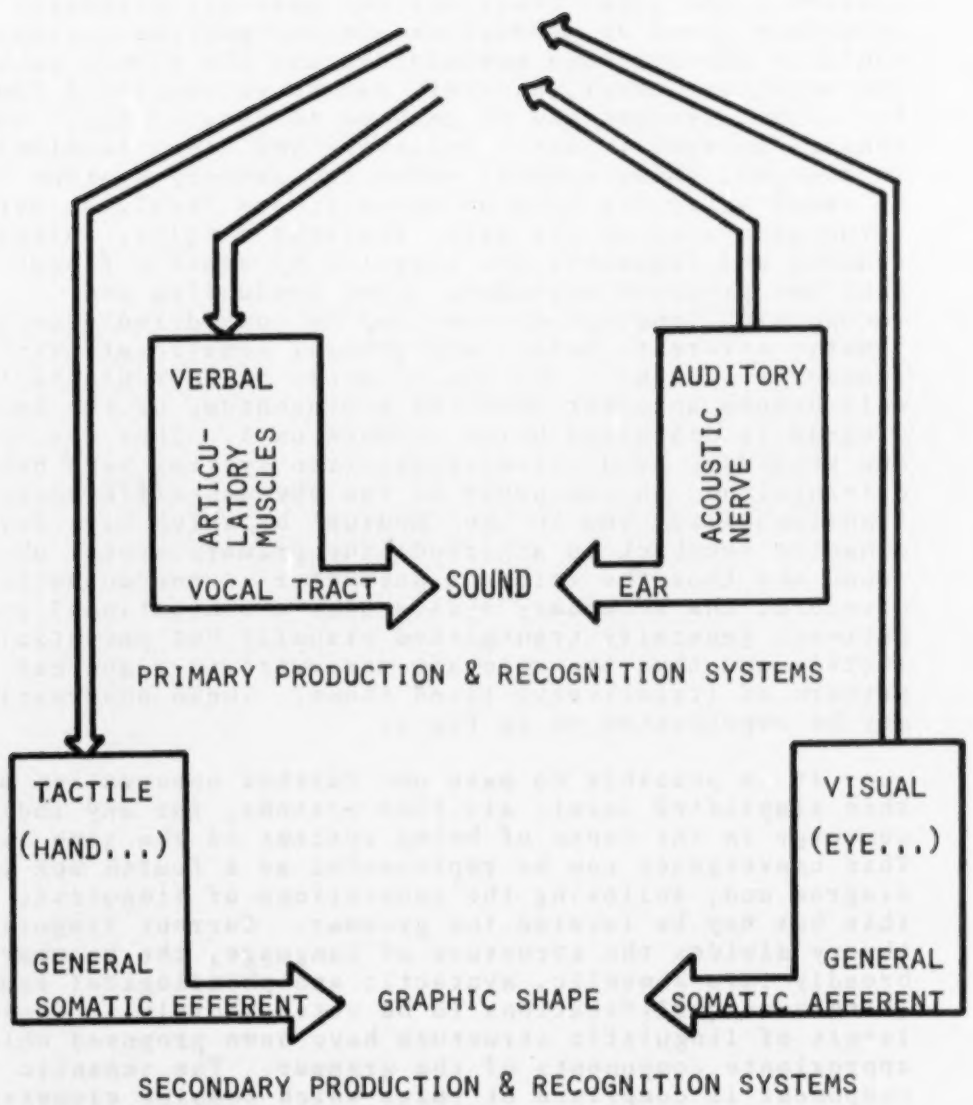

Fig.1 The Peripheral Language System

listening systems. The derivative nature of reading and writing has been pointed out by Azcoaga [1970]. One might propose a primary level of production and recognition systems - the vocal tract and the auditory pathways; a secondary level of production and recognition systems would be the arm/hand musculature and the visual pathways. The secondary level, however, can be extrapolated from its normal systems and be adapted to general motor and sensory systems in man. Following the usual terminology in anatomy, these general motor and sensory systems should be named after the type of nerve fibers involved; peripheral structures such as the skin, skeletal muscles, joints, tendons and ligaments are supplied by somatic fibers, and thus the proposed secondary level production and recognition language systems may be considered general somatic efferent (motor) and general somatic afferent (sensory) systems. The consequences of this distinction will become apparent when the neuroanatomy of the box-diagram is discussed below in Section 5. Thus far, primary and secondary production/recognition systems have been distinguished on the basis of the obvious differences in transferability and in the 'medium' by which high-level semantic feedback is achieved: the primary system uses sound and thus the relevant parameter is the acoustic waveform, the secondary system uses a conventional graphic pattern, generally transmitted visually but potentially tactile and thus the relevant parameter is a general pattern of (relatively) fixed shape. These observations may be represented as in Fig.1.

It is possible to make one further observation at this simplified level: all four systems, for any individual converge in the sense of being systems of the same language This convergence can be represented as a fourth box in the diagram and, following the conventions of linguistic theory this box may be labeled the grammar. Current linguistic theory divides the structure of language, the grammar, broadly into semantic, syntactic and phonological aspects (with some qualifications to be noted shortly). Various levels of linguistic structure have been proposed which approximate components of the grammar. The semantic component is comprised of rules which combine elements of meaning into readings for phrases and sentences; the rules are defined across syntactic constructions produced by the base rules of the syntactic component. The syntactic component is comprised of two sets of rules: (1) base rules which define the structure of phrases within a sentence and which may be considered a universal set for all languages from which a sub-set is drawn for each language and (2) transformational rules, a set of operations which manipulate the structures specified by the base rules in

order to approximate the derived syntactic structure associated with actual sentences of a language. There is presumably a universal set of transformational operations, too, from which are taken the specific set for a language. The phonological component is comprised of a set of rules which convert an abstract generalized representation of the sound patterns of a language into a systematic phonetic representation; there are attempts to relate this output to the observed articulatory behavior (and therefore the acoustic waveform) of speakers of the language. The lexical component contains the elementary units which the above sets of rules operate upon or manipulate. These units are identified as features: an appropriate set of semantic, syntactic and phonological features defines a lexical entry (morpheme, or 'word'). In some current conceptions of the grammar, the semantic and syntactic components are not thought to be separate but rather as a unified body of rules. This would remove the level of deep structure, (the syntactic construction specified by the base rules, upon which both semantic rules and transformations operate - the former to interpret meaning and the latter to generate surface structures) from the model. At present the evidence from aphasia which might be considered as bearing on this last question is equivocal although tending to support the former (standard) model of separate components. With this reservation in mind, the two components will *not* be shown as separate in the model, since the intricacies of the aphasic data are beyond the scope of this proposal. Some discussion can be found in Whitaker [1969, 1970, to appear] and an alternative interpretation in Weigl and Bierwisch [1970]. The grammar to be modelled is comprised of these three components: the phonological system, the syntactic/semantic system and the lexicon. The basis of this proposal is in linguistic theory where with the exception of the separation of syntax and semantics, reasonably good motivation is found for the individual components. The grammar will integrate information that goes to and comes from the four systems postulated before, in the sense of pairing meaning with sound for production and pairing sound with meaning for recognition. For a more adequate linguistic description of the general properties of a grammar see Postal [1968].

It was argued before that linguistic ability should be distinct from abilities such as problem solving, cognition, the 'abstract attitude' (or introspective ability) and the like. This suggestion can be made more specific now by proposing that such other abilities that affect but are not the same as the linguistic ability must be represented in the box-diagram as separate systems.

The problem solving ability and general cognitive functions may not be behaviorally or neurologically distinguishable from each other at present, so they will be lumped in the model as a general cognitive system - cognition. The nature of memory *per se* is also neurologically elusive, although the various forms of amnesia strongly indicate that it is a separate system from the language system regardless of whether or not it can be anatomically localized; notice however that the memory-effector system, by which is meant the system which executes memory storage, may be considered separately from the storage proper (see Talland [1965] for further details). Emotions and attention, which obviously affect the operation of the language system, are intuitively less closely related to it in terms of rules, units and components than the other systems proposed; aside from providing a box for attention, for reasons to be developed in Section 5, no further treatment of these systems will be undertaken. An extended discussion of them may be found in Grossman [1967]. The components that have been suggested so far, based in part on intuition and in part on evidence from linguistics and psychology, represent in a simplified way the major components of the language mechanism as a whole and some systems which are closely related to it. These proposals are schematically shown in Fig.2.

There are some logical implications of this schematic of the language system which ought to be pointed out. A person can manually copy a visually presented pattern either directly in response to the pattern or indirectly from a memory trace of the pattern; this can be done regardless of whether or not the pattern has any meaning to the person. It is of interest to note that brain lesions can impair either the direct or the indirect pattern-copying ability. A meaningless sequence of letters or a word in an unfamiliar writing system can be copied equally well without invoking any of the mechanisms in the language system. Therefore, there is a route connecting the peripheral visual system with the peripheral tactile system which is not mediated by the grammar but by a visuo-spacial system, as shown in Fig.2. In the simplest examples it is obvious that such a system and pathway must exist. It is not clear from the neuropsychological and anatomical evidence that this system is completely independent of the language system or cognitive systems in general. A good review of the problem with references for detailed study may be found in Benton [1969]. The model presented in Fig.2 does assume a separation of the systems, as is shown. The four peripheral language modalities converge on a common grammar, as previously noted. Notice that the alternative to this view would be a 'grammar' for each

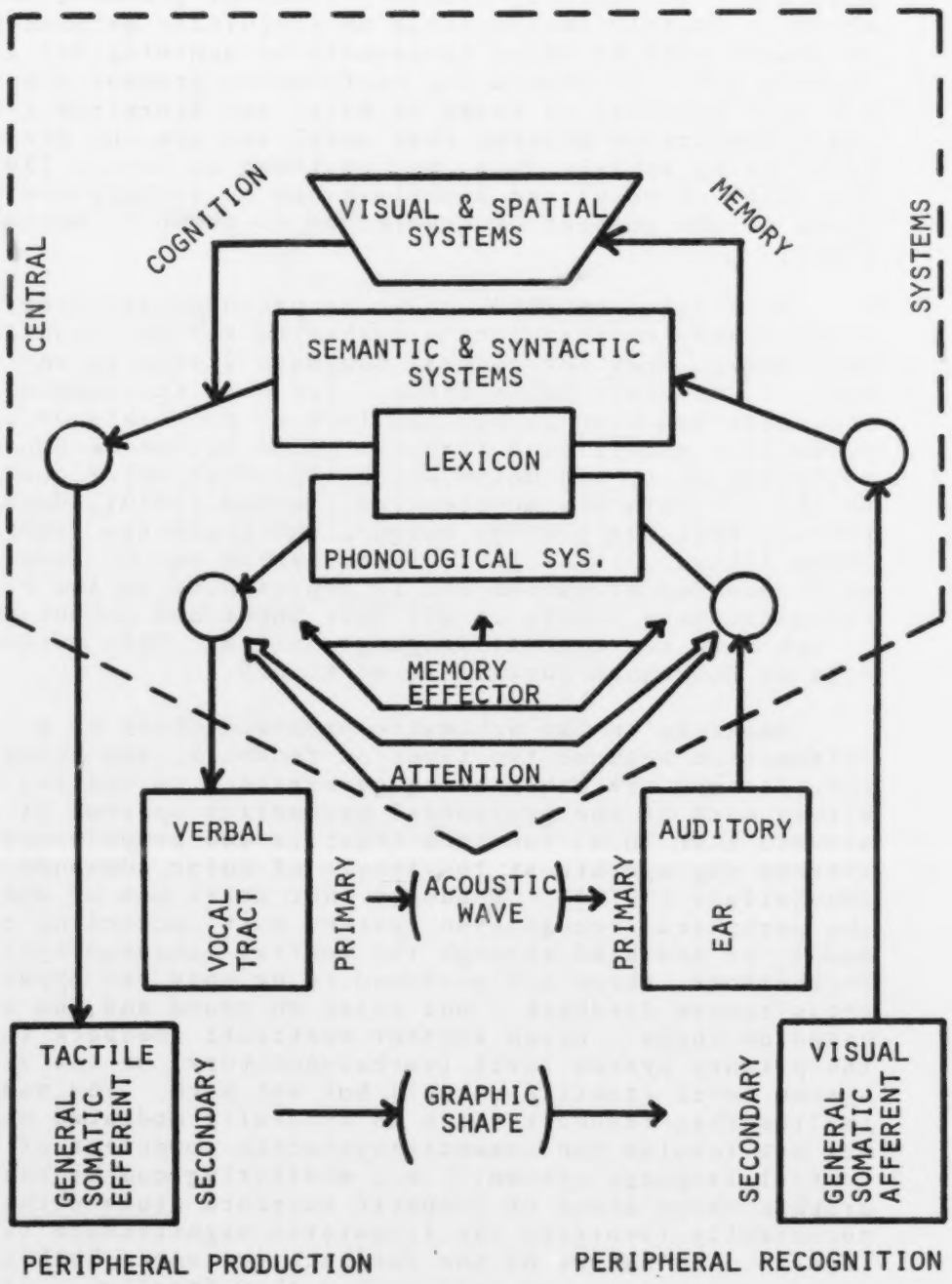

Fig.2 The Language System

modality - an auditory grammar, a verbal grammar, etc. - which is clearly unacceptable on linguistic grounds but of course must be shown inadequate on neurological grounds. Another model incorporating *performance* grammar components for each modality is found in Weigl and Bierwisch [1970] and a comparison between that model and the one proposed here, using aphasic data, may be found in Traill [1970]. The model of Weigl and Bierwisch can be re-interpreted in terms of the present framework, as is shown in Whitaker [1969].

Regardless of which model is presupposed, however, it is necessary to postulate a mechanism for the transfer of information from the central language system to any of the modality-specific sub-systems. The need for such a mechanism has been recognized in many proposals of speech production models as a transfer point between linguistically motivated units and units which represent motor commands to the articulatory muscles (cf. Tatham [1970], MacKay [1970], Whitaker [1970], Lecours and Lhermitte [1969], Green [1969a], *et al.*). This mechanism may be identified as a *tracking* mechanism and is represented in the Fig.2 schematic as a circle in all four input and output pathways to and from the central language system. This mechanism will be discussed further in Section 5.

Implicit in the schematic proposed above is a distinction between two types of feedback, one being system-specific and the other being generalized to central systems. Within each of the peripheral production systems it is assumed that local feedback (tactile and proprioceptive) systems *may* operate at low levels of motor commands (MacNeilage [1970]). Feedback that makes use of one of the peripheral recognition systems must, according to the model, be mediated through the central language system. Furthermore, there are presumed to be only two types of cross-system feedback - one based on sound and the other based on shape - which further restricts feedback to either the primary system level (verbal-auditory) or the secondary system level (tactile-visual) but not both. The model also implies that feedback which is centrally mediated may or may not involve the semantic-syntactic component of the central language system; i.e., monitoring can be based on graphic shape alone or acoustic waveform alone without necessarily involving the linguistic significance of the signal. The nature of the feedback systems - both their putative existence as well as how they function - is quite open to debate (except for the obvious high-level semantic feedback noted above) at present and the claims just made might be too restrictive to adequately account for the requirements of orderly meaningful language expression.

4. Functional Anatomy of the Central Language System

The remarks in the next two sections on the functional anatomy of the parts of the language system must be considered in light of the following caveats. Any model should be an attempt to integrate some good ideas with some blatantly speculative ones, for the purposes of *initiating* further study not stultifying it. As expressed by Green [1970], 'the search for models is thus not designed to sum up but to define areas of fruitful investigation' (220). The speculative nature of the first three sections should have been obvious; it may appear that the next two sections are less speculative because specific anatomical structures of the central and peripheral nervous systems are named. However, even though the neuroanatomy *per se* is on relatively secure grounds, the hypotheses about the functions of the brain areas under consideration are quite speculative. There are no deliberate distortions but the mere attempt to provide a cohesive model for neurolinguistics will perforce introduce hypotheses for which there is little hard evidence.

There have been few attempts to construct a neurolinguistic model of language, fewer still based on or constrained by anatomical considerations. Green [1969b] has a good review of the models developed so far. As he points out, only the models of Luria [1966] and the Boston Veterans Administration Hospital (BVAH) group (of which Green is a member) are rich enough in hypotheses as well as detailed enough anatomically to be seriously correlated with linguistic theory. The model presented below quite freely borrows from Luria and the BVAH group - they should be consulted directly for much of the clinical data from aphasia studies presented here. There are, in the author's

TYPE OF APHASIA	SITE OF LESION	SPONTANEOUS SPEECH	COMPRE-HENSION	REPETITION	NAMING
Broca's Aphasia	Posterior Inferior Frontal	Non-fluent	Intact	Limited	Limited
Wernicke's Aphasia	Posterior Superior Temporal	Fluent	Impaired	Impaired	Impaired
Conduction Aphasia	Arcuate Fasciculus	Fluent	Intact	Impaired	Impaired
Isolation Syndrome	Association Cortex	Fluent Echolalic	Impaired	Intact	Impaired
Anomic Aphasia	Angular Gyrus	Fluent	Intact	Intact	Impaired

Fig.3 Aphasia Classification by Syndromes, from Green [1969]

opinion, some limitations to these earlier models which collectively justify yet another speculation. Neither model is correlated with contemporary linguistic theory, the particular purpose of the present model. Neither model develops the relationships between central and peripheral mechanisms, a relationship considered important here because of the requirement that the model relate to work in linguistics and therefore work in phonetics. In this regard the work of Darley and co-workers at the Mayo Clinic has been particularly invaluable. Neither model mentions the possible role of thalamic and other sub-cortical nuclei in the central language system, a hypothesis first advanced by Penfield and Roberts [1959] and recently strongly supported by work done at the NIH in Bethesda. And last, neither model attempts to state information flow patterns, a point which is less important than the previous three in some respects since such patterns can in fact be inferred from their models. The model presented below should not be regarded as rejecting the proposals made by Luria or the BVAH group, nor as offering counter-proposals; rather it should be regarded as an attempt to build upon their theories.

Green proposes five aspects of the language ability which may be differentiated in aphasia; he does not imply that there are *only* five aspects, as there obviously are not, but that these are the aspects which the BVAH model emphasizes. These are represented in the following table from Green [1969b], (p.47), as Fig.3. In the main the areas delimited in this model are accepted, but as will be apparent, the interpretations placed upon them with respect to linguistic functions will be rather different.

It is not possible here to adequately depict in diagrams the brain and its components in any really revealing way; nevertheless, at least a gross reference point can be given for the cortical components of the proposed central language system. Aside from these reference diagrams the remainder of the proposed systems in this and the next section will be presented schematically. The cerebral cortex is a sheet of neurons, cytoarchitecturally separable into six layers generally, folded over on itself. The smooth top parts of the folds are called gyri, the creases, sulci or fissures. No two brains are alike in the topographical configurations of gyri and sulci, although usually a number of 'landmarks' can be identified: the Fissure of Rolando which separates the primary motor (anterior) cortex from the primary sensory (posterior) cortex; the Fissure of Sylvius which marks the dorsal (top) edge of the temporal lobe; the major gyri (convolutions) of the frontal and temporal lobes - superior,

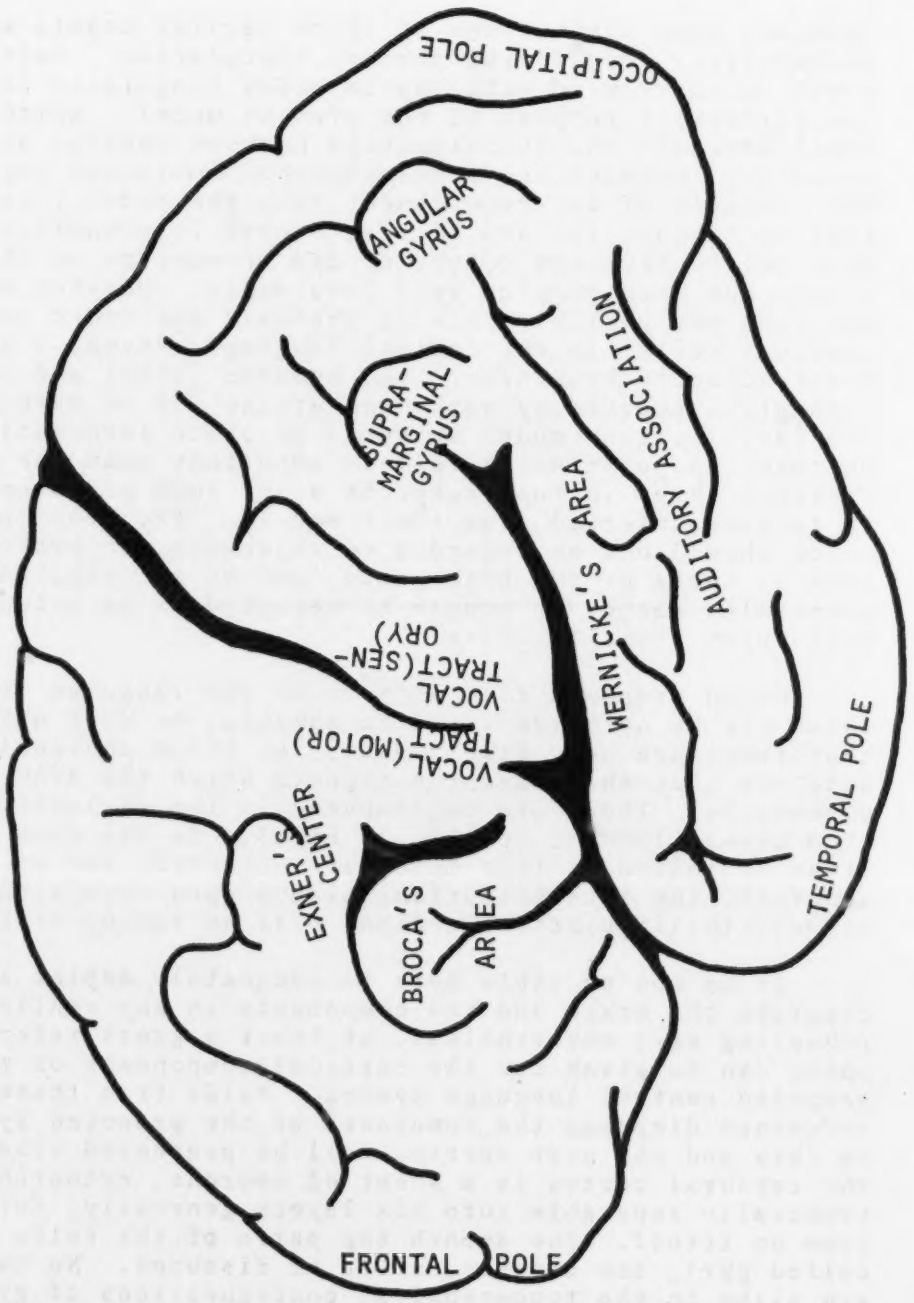

Fig.4 Lateral view of left hemisphere of brain

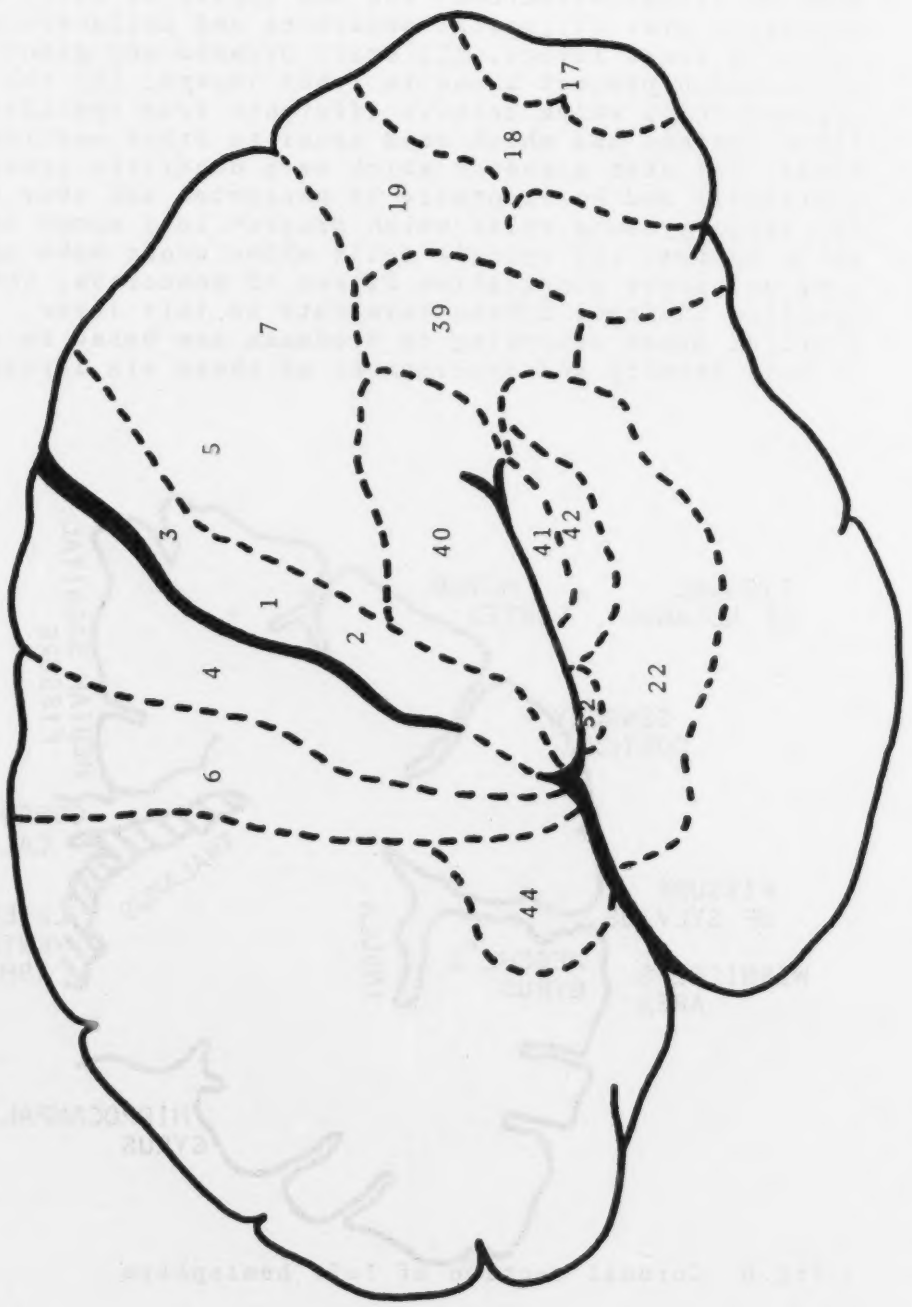

Fig.5 Brodmann areas of left hemisphere.

NOTE ON CYTOARCHITECTURE: The six layers of cells in neocortex are: (1) apical dendrites and collaterals from cells in lower layers, (2) small pyramid and granular cells which project axons to lower layers, (3) the large pyramid cells which receive afferents from specific fibre systems and which send axons to other cortical areas, (4) star pyramids which have dendritic processes vertically and horizontally in neocortex and star cells, (5) large pyramid cells which project long axons into white matter, (6) spindle cells whose axons make up the long and short association fibres of neocortex; the non-specific thalamic fibres terminate in this layer. The cortical areas according to Brodmann are based on variations in cell density and development of these six layers.

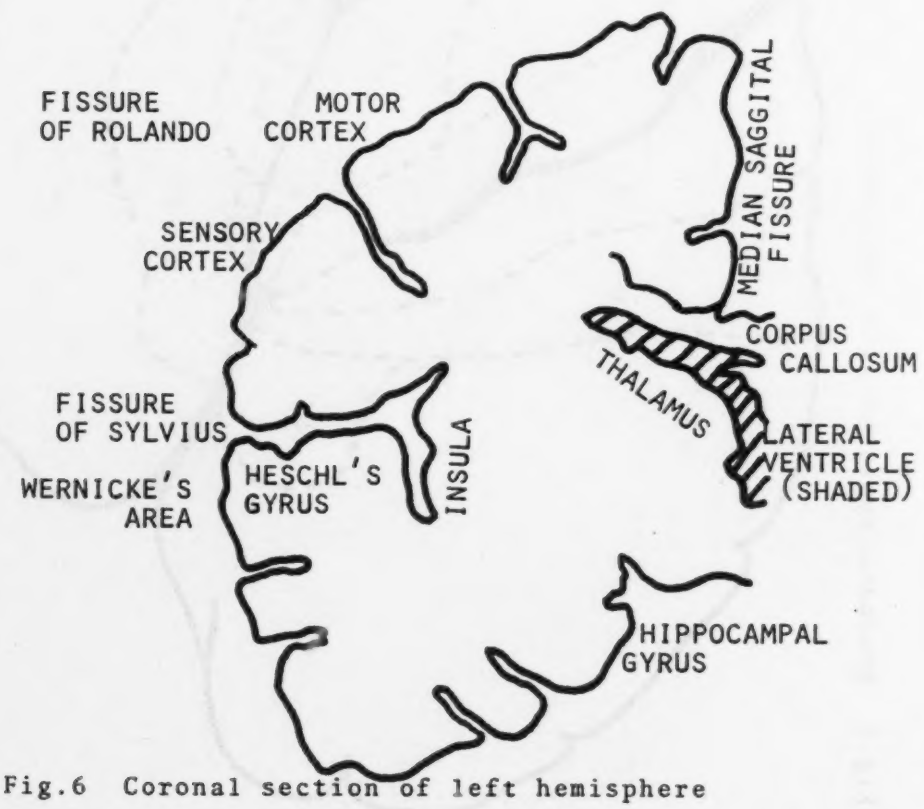

Fig.6 Coronal section of left hemisphere

middle and inferior, proceeding from top to bottom. A simplified sketch of the surface of the left hemisphere, showing the classical names for those parts of the cortex usually associated with language and speech is shown in Fig.4. Since the gross topography alone is quite unsatisfactory as an anatomical basis of the neurolinguistic model, a sketch of the major cytoarchitectural distinctions based upon the original drawings of Brodmann and employing his numbering system, is provided in Fig.5. If a cut is made vertically through Wernicke's Area and the brain is viewed 'head-on' (a coronal section), the resulting view is shown as Fig.6 - the area of importance in this view is Heschl's Gyrus.

What is of interest to note is the remarkable degree to which distinct cellular arrays in the cortex (the Brodmann areas) correspond to the 'areas' classically associated with the language system, a correlation that is easily seen by comparing Figs.4 and 5. Thus Area 44 is correlated with Broca's Area, the foot of the inferior frontal gyrus. A portion of Area 6 is correlated with Exner's Center, the foot of the middle frontal gyrus. The primary motor and sensory cortical areas, anterior to and posterior to the Fissure of Rolando respectively, correlate with Areas 4 and 2, respectively - note that only the 'vocal-tract' areas are shown on the diagram, to indicate that the muscles of the neck and head are represented in those cortical fields. Muscles for the hand, arm, body and leg are represented in ascending order in the motor cortex. The corresponding somatosensory representations are in the sensory cortex. The supramarginal gyrus correlates with parts of Areas 40 and 22, the angular gyrus, with Area 39. Wernike's Area correlates with Area 42 and probably parts of Area 22, and the Auditory Association field with the main part of Area 22. Heschl's gyrus, shown in Fig.6, correlates with Area 41 of Brodmann. Since the architectonic fields just described are essentially the same for all brains and the topography is not, it is reasonable to suggest that these fields are more appropriately considered the anatomical correlates of the language system; in order to simplify discussion however the more familiar classical names will be used throughout this proposal.

As is well-known, cortical fields interconnect with each other (cortico-cortical fibers) as well as with subcortical and peripheral structures. Some interesting conclusions may be made from a knowledge of the connections combined with the behavioral evidence (the apraxias, agnosias and aphasias) due to lesions. First, the central language system (CLS) may be distinguished from its points of contact or association with the peripheral language modalities. It is postulated that the following regions

of the cortex subserve the semantic/syntactic component and the lexicon of the grammar: part of Wernicke's Area, the Auditory Association cortex, the Supramarginal Gyrus and part of the Angular Gyrus. It is highly unlikely that there are strict boundaries to this system in any one brain even on cytoarchitectural grounds, even though we may presume that the general demarcation is definable. One suspects that there are individual differences in terms of how much of these cortical fields are actually recruited for the CLS just as there are individual differences in everything else. Lesions to these areas produce a range of defects that typically involve the following linguistic concepts: selection of appropriate grammatical categories, the syntactic integrity of the sentence as well as its major constituents the noun phrase and the verb phrase, the selection of specific items within a semantic field, the ability to relate sentences of the same meaning to each other using syntactic transformations, the ability to make use of a normal vocabulary range, and the ability to string together meaningful sequences of words. The full linguistic details of these deficits are remarkable confirmation for many of our notions of grammatical organization; some details of these deficits may be found in Geschwind [1967a], Marshall and Newcombe [1966] and in work of the author, and consequently will not be elaborated further here.

The connections of these postulated areas of the CLS satisfy the requirements of the general schematic proposed in Section 3. In particular, each of the areas are connected to each other *via* the short cortico-cortical fibers that are found within the cortical mantle. These fibers, incidentally, rarely are found outside the grey area; the connecting fibers found in the white matter beneath the cortical mantle are generally longer, trans-hemispheric or interhemispheric connections. It is the latter fibers that connect some of these areas of the CLS with the peripheral production modalities. Considering both types of connecting fibers, we may conclude that the following pathways for the transfer of signals in the CLS exist: the Angular Gyrus connects with visual association cortex, Brodmann Area 19; the Supramarginal Gyrus connects with general somatic afferent association cortex in Areas 40 and 7; the posterior portion of Wernicke's Area and the Auditory Association cortex connect with the association cortex (Area 42) surrounding Heschl's Gyrus, and portions of Wernicke's Area, Auditory Association cortex and Angular Gyrus, connect *via* the long inter-cortical fibers known as the Arcuate Fasciculus with Broca's Area and Exner's Area.

Thus the anatomical connections provide the grammar or CLS with the requisite sources of information and outlets for expression that a logical schematic requires. Geschwind [1965] discusses these connections in greater detail and in addition develops postulates for the role of interhemispheric connections such as the corpus callosum, and for the role of sub-cortical connections to the limbic system. He also notes some interesting properties of the cortical areas proposed in the CLS mechanism. The myelination of the axons within and leaving the infero-parietal region (Supramarginal and Angular Gyri) is completed much later than in other cortical areas. The myelin sheath surrounding an axon presumably acts as an 'insulator' that permits neuronal spike trains to travel at a much faster rate. Dendritic growth is completed later in this region than in other areas of the cortex, too. The dendrite complex of a neuron may be regarded informally as its information source: the more complex a dendritic structure, the more sources of excitatory and inhibitory impulses available to influence the firing of the cell body (soma). It is of course suggestive that this region should at the same time be well-correlated with the acquisition of language in the child. There are few homologies between the infero-parietal region in man and any region in the brain of other primates - i.e., a region which seems to sit squarely at the junction of the parietal (somatosensory, spatial, etc.), occipital (visual, etc.) and temporal lobes (audition, etc.). The interconnections established by the infero-parietal region led Geschwind to postulate that this area was a pre-requisite for the capacity of language in man. There are, to be sure, questions remaining about the neuroanatomy of this region as well as the putative lack of correlation with other primate brains, but in the main this hypothesis appears to be a remarkable insight that takes full advantage of the relevance of neuroanatomy to an understanding of language.

The thalamus, located approximately in the center of the brain in the area which would be the topmost ramification of the brain stem, has not been discussed in great detail in most recent work. It was originally proposed by Penfield and Roberts [1959] to be part of the CLS, even though they had quite weak supporting evidence. It now appears that to a degree they were correct. It is possible, using electro-stimulation techniques (as a concomitant of neurosurgery) to cause aphasic language disruptions in the *left* pulvinar and perhaps in some other left-side thalamic nuclei. The fact that the right pulvinar does not yield such data is rather dramatic corroboration of the role of these thalamic nuclei in the CLS, since the lateralization of cortical language functions has been well established. A full discussion of the

thalamic evidence may be found in Ojemann, Fedio and VanBuren [1968]. Connections between the pulvinar and those parts of the cortex known to be part of the CLS, in particular the Supramarginal and Angular Gyri (infero-parietal cortex), are quite well established as ascending fibers (thalamo-cortical) but slightly less well established as descending fibers (cortico-thalamic). On the aphasic evidence alone it seems necessary to include the pulvinar in the CLS mechanisms, even though fuller details are needed.

In a very interesting case history studied by Geschwind, Quadfasel and Segarra [1968], a sub-part of the CLS structures was preserved. They observed that most of the cortex was destroyed by CO poisoning; however,

> ... a detailed study of serial whole-brain sections showed intactness of auditory pathways up to and including Heschl's gyrus, of Wernicke's area and Broca's area and of the arcuate fasciculus connecting these two cortical regions, of the lower Rolandic cortex and of corresponding portions of the pyramidal tract. The hippocampal region (except for Sommer's sector) and the structures of the limbic system were well preserved, as was the reticular substance of the brain stem. (327)

The patient, although unable to make use of the semantic/syntactic component and the general cognitive system, was able to repeat verbal material very well, to complete well-learned stereotyped phrases (about which more will be said shortly) and engage in a limited verbal learning of new songs played on the radio. As an exemplary case supporting the localizationist position adopted here it is well worth noting; however it has, for linguistic theory, an even more interesting concomitant. It is patently obvious that the verbal behavior of this patient required an intact phonological component of the grammar. In fact, it is highly probable that the patient's verbal behavior can be simply characterized as a separation (disconnection, to use Geschwind's terminology) of the phonological component from the remainder of the CLS and from other related systems. Including this evidence and that pertaining to the pulvinar, one could suggest that the schematic in Fig.7 adequately represents the neuroanatomy of major structures of the central language system. The three part structure incorporating Heschl's Gyrus, some of Wernicke's Area, the arcuate fasciculus, Broca's Area

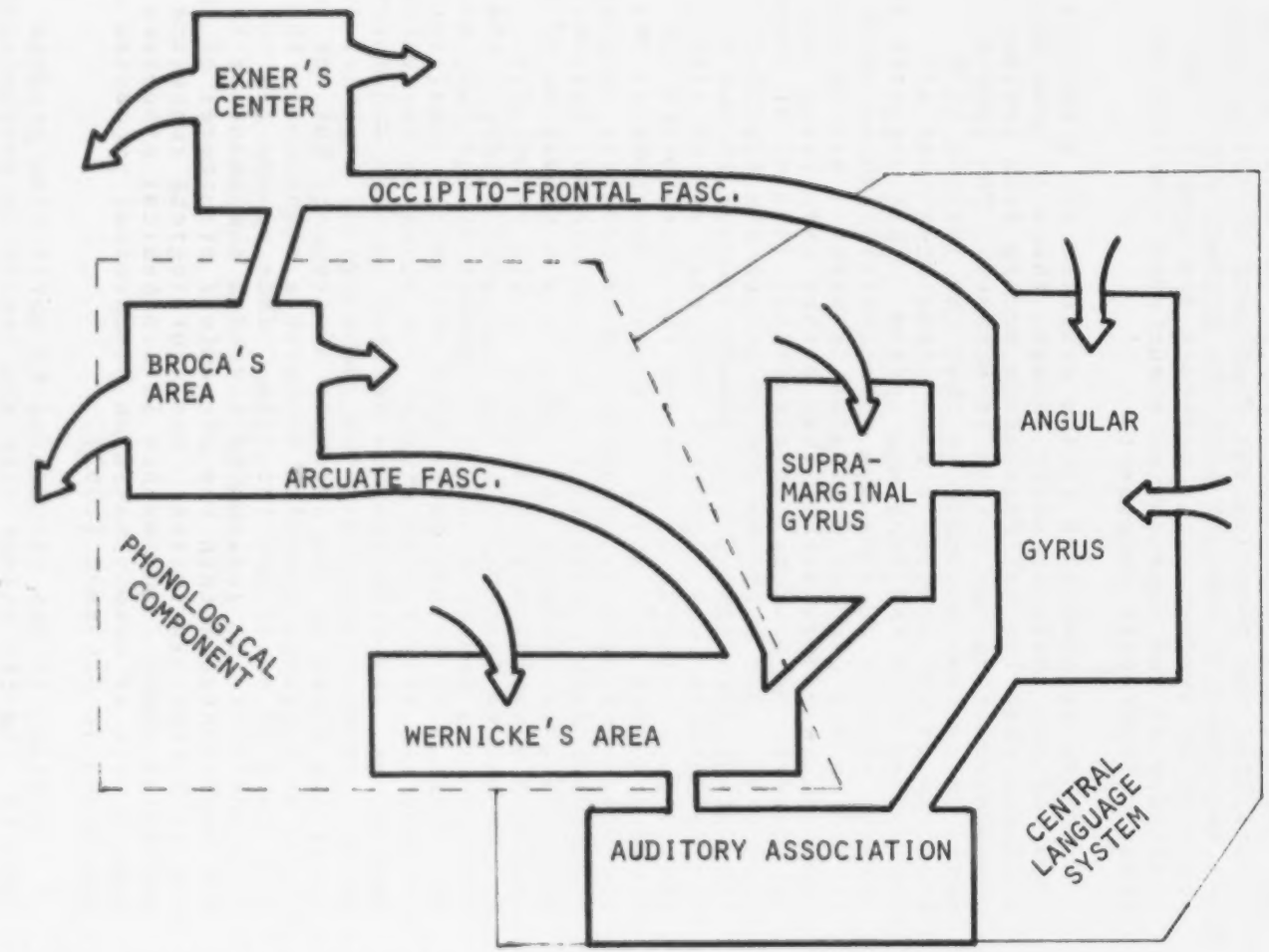

Fig.7 Anatomical schematic of the central language system

and perhaps the vocal tract area of the motor cortex, constitutes the phonological component of the grammar and its interface or contact with the primary production and recognition systems - the auditory and verbal. The remainder of the represented structures comprise the semantic/syntactic component.

There is presently little evidence for a cortical locus of the lexicon; oddly enough, there is even less evidence that lexical disruption occurs from lesions in *non-central* language system structures. The probable view based on current evidence is that the lexicon is a property of all nervous system structures that are associated with the language system. This suggests that certain aspects of language, particularly those which are stored or are part of a linguistic memory, may be biochemical in nature rather than either structural (areas or networks of neurons) or electrical (trains of neuronal impulses). One might be led to accept the hypothesis of Longuet-Higgins [1968] that memory storage in the brain is 'holographic'. One expects a reduction in the clarity and detail of a hologram if parts of the photo plate are destroyed, but not 'holes' or missing portions of the picture. Analogously, destruction of cortical language structures does not destroy a lexicon *per se*, but only quantitatively reduces it - this is a concomitant of the aphasias associated with lesions to CLS structures. Notice though that there is in fact good evidence that the lexicon is a separate neural component of the CLS, even if the separation is not based upon anatomical localization. Lesions can selectively disrupt the rules of derivational morphology, the relationship between content and function words, semantic features, the phonological organization of words (in terms of the sequence of segments but not in terms of language-specific acceptable sequences), the referents of words and the like. Thus we are led to conclude that the lexicon is indeed a component of the CLS, but represented within the anatomical structures of the CLS not as a separate system. One neurological correlate which satisfies these requirements is biochemical processes. Some models of memory based on biochemical processes are discussed in Grossman [1967].

Lesions in CLS structures by definition produce aphasia. To the extent that the lesion is preponderantly 'towards' the contact point between the CLS and one of the peripheral modalities, there will be an increasingly greater component of apraxia (if near a production modality) or agnosia (if near a recognition modality). Otherwise, the deficit will be manifest in all language modalities to some degree. Lesion affecting systems related to but distinct

from the CLS cannot properly be said to cause aphasia, though there may be aphasic symptoms in conjunction with them. These related systems were represented in Fig.2 and may be briefly mentioned again in anatomical terms. It is presumed that the major structures correlated with emotions comprise the limbic system which would influence the CLS through either the frontal lobes or thalamus. The limbic system, of course, is quite similar in man and other animals and a great many homologies may be drawn. The Reticular Activating System is usually associated with the control of attention in the nervous system, following the work of Magoun [1963] and others. Although memory storage appears to have no specific locus but is a general property of nervous tissue, there is good reason to assume that the momory-effector system is anatomically well-defined. Based upon much evidence that lesions in these areas impair or destroy the ability to store new memories, we may assume the memory-effector system is comprised of the mamillary-hippocampal, thalamic system outlined in, e.g. Talland [1965] and others. The visuo-spatial system, to the extent that it is separable from the CLS, probably is subserved by the parietal lobe of the *non-dominant* hemisphere (which is usually the right hemisphere). This suggestion is common to many studies of constructional apraxia and other visual-spatial orientation disorders directly associated with the ability to manipulate patterns without verbal mediation. Benton [1969] has a review of this literature.

To conclude this section on the CLS, brief mention should be made of the matter of hemispheric asymmetry. The evidence that for most people the CLS is strictly lateralized in the left hemisphere is statistically overwhelming. Some data indicating that the isolated non-dominant (right) hemisphere can process language (where isolation is accomplished by surgical section of commisural connections - Gazzaniga [1970]) in all likelihood reflects the fact that some aspects of linguistic structure are expressed in the general somatic afferent and efferent properties of the secondary peripheral systems; it could be predicted that certain parts of the non-dominant hemisphere may have stored or retained some control over, these general neuronal properties. The non-dominant hemisphere control of language so far has been shown to be limited to a small subset of concrete nouns - those limited to familiar household objects. There is also the possibility, of course, that in some brains the lateralization tendency is incompletely realized. With these qualifications, the CLS mechanisms are presumed to be found only in one hemisphere, usually the left.

5. Functional Anatomy of the Peripheral Language Modalities

The overriding principle of the four peripheral language modalities, a principle not generally emphasized in neurolinguistic models, is the distinction between modality-specific linguistic functions and the physiology and anatomy of the system itself. These will be referred to as modality-specific and system functions in the following text. The importance of the distinction is well understood by neurologists and speech pathologists who must differentially diagnose, for example, a right visual field hemianopia from alexia - i.e. a visual loss or blindness must be distinguished from a deficit in the ability to process written language materials. Analogous distinctions can be made in all the peripheral modalities. It will be useful to establish the terminology used to characterize this principle, as employed here. ALEXIA will be used to refer to a modality-specific linguistic deficit in the visual system. AGNOSIA will be used to refer to a modality-specific linguistic deficit in the auditory system. In the production systems, AGRAPHIA will refer to a writing deficit that is independent of muscular control of the arm and hand and APRAXIA will refer to a comparable deficit in the linguistic control of the vocal-tract musculature. In reference to production systems in general, the term apraxia may be used to refer to general linguistic impairments; the term agnosia will be similarly used to refer to recognition systems in general. DYSARTHRIA is a term reserved for system dysfunctions in the verbal language modality, i.e. loss or impairment of control of the muscles of the vocal tract for any purposes rather than just linguistic. This usage does not completely coincide with the terminology found in the aphasiology literature;

there are several reasons for the discrepancies. Clinically it is useful to distinguish some sub-types of deficit that are often associated with aphasia but may not be due to damage of the central language system or any peripheral language modality; for example, deficits in the patient's ability to process and manipulate visuo-spatial pattern information probably has an anatomical locus in the parietal region. Some types of this disorder are more frequently located in the right (non-dominant) parietal lobe and others seem to be more frequently associated with left parietal lesions. This deficit has been labeled by many researchers 'constructional apraxia' (see Benton [1969]) although from the point of view taken here it is difficult to see why it should be linked to production deficits rather than recognition deficits. There is also in the clinical literature a number of terms that are used to attempt to break down a general deficit into its linguistically relevant syndromes. Thus one finds terms such as pure word deafness, sensory amusia, transcortical sensory aphasia, auditory agnosia, etc. And last, one often finds in the literature a number of terms referring to the same deficit: Broca's Aphasia, motor aphasia, apraxia of speech, cortical dysarthria, and the like (Darley [1968]). It would be impossible as well as pointless to make any attempt to sort out the terminological issues in aphasiology in this paper. Some sources to refer to for studies of this kind are Nielsen [1946] which is a bit out of date but very complete, or Bay [1967]. References to components of dysfunctions in this proposal will be made in linguistic terminology wherever possible; hopefully this framework will obviate the need for an elaborate clinically-based classification system.

5(a). <u>The Visual Language Modality</u>

Details of the visual system of man are readily available in most texts of neuroanatomy and physiological psychology; therefore the specifics of this system will be considered only in bare outline. The gross anatomy of the visual pathways is diagrammed in Fig.8, and includes its presumed terminus as a language modality in the region of the Angular Gyrus in the left hemisphere. The principal optic characteristics are well known: the right half of the visual field is focussed on the left half of each retina, and *vice versa*. Each optic nerve carries a right and a left half-field (divided vertically) to the optic chiasma where the pathways carrying left visual field information (from the right retinas) converge in the optic tract of

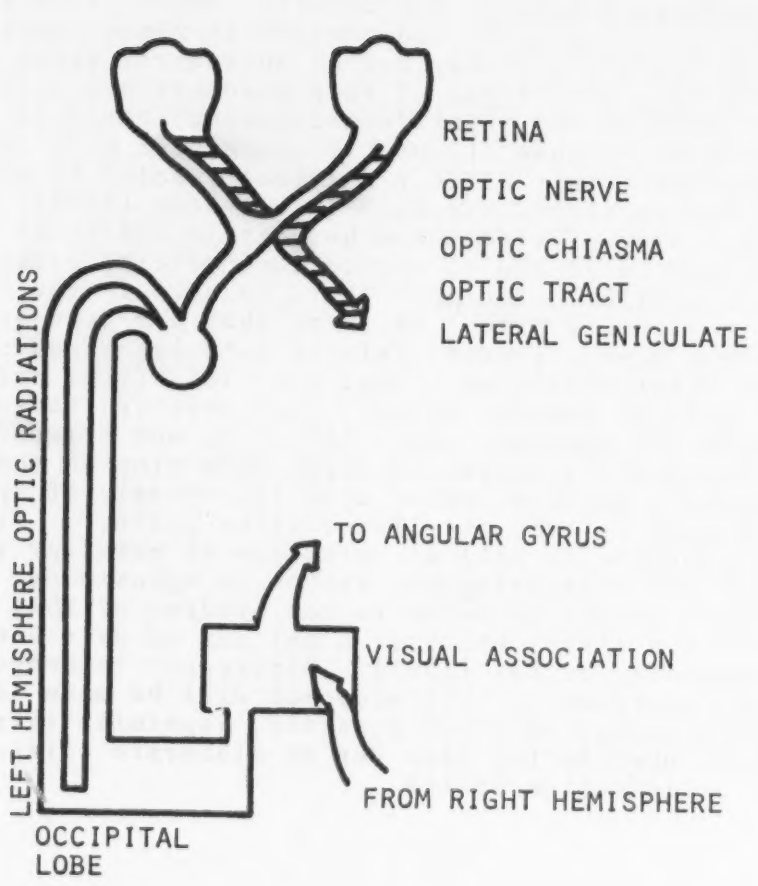

Fig.8 Anatomical schematic of the visual modality

the right hemisphere; right visual field information proceeds in the optic tract of the left hemisphere. At the lateral geniculate bodies posterior to the thalamus, the optic tract divides into an upper and a lower quadrant - the optic radiations. The two optic radiations proceed through the hemisphere to the primary visual cortex (occipital lobe) which is Area 17 of Brodmann. In the right hemisphere, for example, the lower left quadrant of the visual field projects to the cortex anterior to the calcarine fissure and the upper left quadrant projects to the cortex posterior to the calcarine fissure at the occipital pole. The reverse, of course, holds for the left hemisphere. From Area 17 visual information is presumably processed through the visual association cortex (Areas 18 and 19) and then to the Angular Gyrus and other temporo-parietal areas. Information from the right occipital lobe (information pertaining to the upper and lower quadrants of the left visual field) is not directly exchanged with the left occipital lobe until it reaches the visual association cortex; primary visual cortex projects to secondary visual (association) cortex and the latter crosses to the opposing hemisphere *via* the splenium of the corpus callosum. The corpus callosum connects many cortical fields between the two hemisphere, but not (as discussed in Geschwind [1965]) between primary projection cortex; this suggests of course that the interhemispheric information transfer occurs only after some sort of 'analysis' or 'recoding' has occurred. Geschwind has some interesting speculations on this and should be consulted for complete details. Lesions which produce aphasia, i.e. lesions to left hemisphere CLS structures, often affect the optic radiation fiber bundle which passes below the cortical mantle of the temporal lobe. It is common to find therefore, right visual field deficits (since this is the visual field for which information is carried in the left hemispheric optic radiation) which can on occasion be mistaken for alexic difficulties. Specifically, it can happen that a patient will fail to read a word or sentence properly because he does not actually see the print; when the visual field cut happens to divide the subject from the predicate, or a derivational affix from its stem, it is not hard to imagine the erroneous linguistic conclusions that can be made.

Assuming a normal corpus callosum and no damage to the optic system from the retina to the occipital lobe, there is little evidence now for any lateralization (hemispheric dominance) of visual function - there is only the division of right and left visual fields (and quadrants) which are presumed to be equally displayed to each hemisphere. Laterality in the visual system does not appear to be

introduced until the visual patterns are processed in relation to memory storage, probably at the level of the Angular Gyrus; at this point the left hemisphere dominates for written language information, the right for general pattern recognition. Considering the nature of alexic impairments, one or two speculations may be made about the topmost level of the visual language modality. It is possible to distinguish the task of identifying a single written word as opposed to the task of identifying a phrase or sentence. The whole visual language modality can, of course, be 'disconnected' from the CLS, producing complete alexia. And it is possible that different classes (both grammatical and semantic) may be differentially impaired in reading (some interesting discussion of the last possibility is found in Marshall and Newcombe [1966]). From these and other considerations to be mentioned below, information flow in the visual language modality could be postulated as follows: up to the visual association cortex (Areas 18 and 19) the primary function is pattern integration within the specific half-field. The Angular Gyrus however will have patterns 'presented' to it which, because of the connections in the splenium, will be full field; based on the contact the Angular Gyrus has with the CLS, these patterns will be correlated with language storage. Since visual patterns are ordinarily not time-varying, it may be unnecessary to consider such time-based concepts as tracking and guessing strategies but the principle must be very similar. One buffer (from the visual association cortices) has the organized pattern and the other (from the Angular Gyrus) the sources by which the pattern information can be given linguistic significance. Note that saccadic eye movements will in fact project a time-varying image on the retina; it is assumed that this time base subserves system-specific physiological functions and is not related to the transmission of high-level pattern information to the Angular Gyrus. The different types of deficits found in alexia will presumably be associated with either lesions in the Angular Gyrus or in other parts of the CLS and not with lesions in visual association cortex or other parts of the visual system.

It was noted in Section 2 above that the secondary level production and recognition systems could be regarded as general somatic efferent and afferent, respectively. This implies that the reading buffer in the Angular Gyrus which ordinarily makes use of visual input patterns can if necessary compute on patterns from somesthetic association cortex (parietal lobe). Braille reading should make use of a tactile pathway to the primary sensory cortex (posterior to the Fissure of Rolando and

above the area designated as vocal-tract (sensory) in Fig.4); information would pass from this region through the parietal lobe to the Angular Gyrus region. If correct, this model would expect that blind persons who read braille are susceptible to alexic deficits identical to those that other persons may suffer, except that they would be manifested in part in tactile sub-system. The model would also predict that the deaf who use sign language could suffer these deficits and not deficits *typical of apraxia or agraphia*. There is evidence to support these hypotheses. In brief, the Angular Gyrus reading buffer should be capable of computing the linguistic significance of any sensory signal coded as a graphic pattern, although it is obviously most proficient at receiving information from the visual system buffer. The contact point between the putative buffers was represented as a circle in Fig.2 - it is the point at which the guessing mechanisms operate, assuming as in this model that language functions are strategy-governed rather than rule-governed. The Angular Gyrus region is also the contact point through which feedback information is provided to the tactile system for control of writing and typing. This information is probably carried by the occipito-frontal fasciculus and will be discussed again in Section 5(c) below.

5(b). The Auditory Language Modality

Like the visual pathways, the basic anatomy of the auditory paths is relatively well-known and little detail needs to be noted here. The general schematic is shown in Fig.9. Signals from the cochlea (organ of corti) pass along the acoustic nerve (Cranial N. VIII) to the ventral and then dorsal cochlear nuclei located in the medulla just below the brachium pontis. From the ventral cochlear nucleus some fibers proceed to the ipsilateral superior olivary complex and some to the contralateral superior olivary complex; ipsilateral fibers then proceed from each olivary up the brain stem to the inferior colliculi. The remaining fibers which go to the dorsal cochlear nucleus cross the midline to the contralateral superior olivary complex and in combination with other fibers also proceed up the brain stem to the inferior colliculus. Some fibers again cross the midline at the inferior colliculi level; the rest pass to the medial geniculate bodies and then to Heschl's Gyrus. It is obvious therefore that each Heschl's Gyrus receives signals from both ears, apparently in approximately equal dis-

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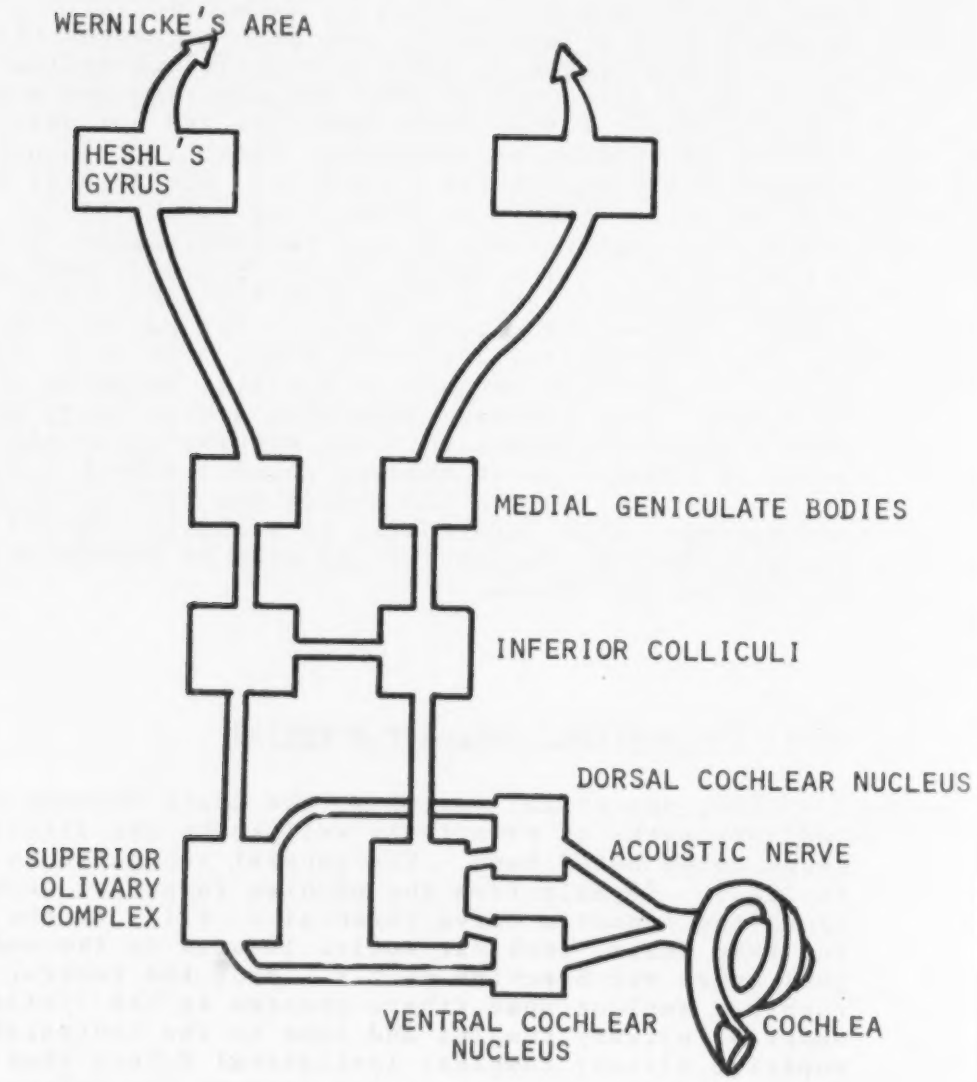
Fig.9 Anatomical schematic of the auditory modality



tribution. The acoustic waveform is probably 'displayed' across Heschl's Gyrus in a frequency array - higher frequencies to the posterior end and lower frequencies to the anterior end near the temporal pole. Presumably the intensity differences in the waveform are also encoded directly into Heschl's Gyrus. Although each Heschl's Gyrus or primary auditory projection cortex (Area 41 of Brodmann) receives virtually identical signals, the signals are clearly not processed identically. There is much evidence - from psychophysical studies as well as lesion studies (reviewed in detail in Vignolo [1969]) - that linguistically significant acoustic signals are preferentially analyzed by the (right ear and) left hemisphere and other kinds of acoustic stimuli are processed by the right hemisphere (e.g., music). It is not certain whether the sorting of linguistic and non-linguistic acoustic signals is achieved prior to their arrival at the cortex (approximately 10msec. after stimulation in the organ of corti) but it is quite certain that the left Heschl's Gyrus is the only one which is part of the CLS. Heschl's Gyrus interconnects only with adjacent cortical fields (Area 42 or Wernicke's Area) but from here information may either follow the arcuate fasciculus fibers directly to Broca's Area - the pathways suggested earlier as the basis for fast phonological but non-semantic feedback to the verbal system and therefore the primary anatomical substrate from the phonological component of the CLS - or, it may be further integrated by CLS structures connected to Wernicke's Area - the Auditory Association cortex, Supramarginal Gyrus, etc.

Like the visual system's adaptation for language the auditory system must also provide a buffer for a coding of the acoustic waveform that allows the auditory control to match linguistically significant storage to incoming signals. It is likely that this proceeds in two steps: the first being a straight phonological reading and the second a semantic/syntactic reading. And, incidentally, it should be suggested that the visual system operates analogously. There is much evidence from the predominantly sensory aphasias to support this view (and a fuller discussion of it may be found in Weigl and Bierwisch [1970]): the ability to repeat but not comprehend or spontaneously speak, an impairment of auditory comprehension with good visual comprehension and spontaneous speech, and similar gross functional isolations. What is of even more interest is that agnosia can be as selective as alexia in affecting semantic class, grammatical categories, phrases and sentential structure; at least, assuming that the judgments and tests of impaired auditory recognition cited in the literature are reasonably accurate and valid, one can make such linguistic interpretations.

The buffer relationship in the auditory modality is quite obviously time-locked, which would lead us to expect that it has optimum quantities of information that it can process at a time as well as an optimum speech of input. It is rather interesting to observe that, in this model, the primary production and recognition systems (verbal and auditory) have time-locked tracking and guessing strategies and furthermore compute on information that is virtually modality specific. The secondary production and recognition systems (tactile and visual) are not so time-locked and the information used is more like general somatic afferent and efferent patterns. An obvious speculation to make is to inquire whether or not these qualitatively different operating mechanisms differ in other respects as well, namely, the degree to which features of input and output information must be hierarchically arranged, the degree to which the primary system must be strategy-governed and the secondary system (possibly) rule-governed, whether all these ideas bear upon questions of acquisition, long-range stability (resistance to linguistic change) and whether there are general constraints on the possible information load that graphic patterns may freely carry (i.e. in a day-to-day situation rather than in the potentials of such things as secret codes or phonetic diacritics). It is remotely possible that the general somatic afferent/efferent aspect of the secondary systems, and the possibility that the central and peripheral nervous system structures which usually carry this information are more removed from limbic system influences than the primary system, both contribute to the specific character of writing systems, the reading process and other dependent functions.

5(c). <u>The Tactile Language Modality</u>

The Production Systems are not as easily incorporated into the neurolinguistic model, partly because the functional neuroanatomy is less well understood, partly because the model here must explicitly account for errors (since errors overtly show up in the data, unlike the situation for the recognition systems) and partly because any coherent speculations may immediately be confronted with the results of the systems in operation - observed verbal/writing behavior. One interesting aspect of production systems is the so-called ictal speech automatisms (see Chase [1967]) which appear to be fully encoded motor programs for words and phrases, generally ones which are emotionally charged such as swearing.

These utterances may be produced by patients with major lesions of the anterior branches of the middle cerebral artery (i.e. with massive destruction of Broca's Area and surrounding vocal-tract motor cortex) who otherwise are unable to speak at all. In Whitaker [1969] a patient was described who could produce the utterance 'What are you going to do right now?' with perfect articulation, intonation and clarity, but who had no other expressive speech. Data such as this has led to models which incorporate a special mechanism for ictal speech automatisms that is presumed to be outside the CLS proper. When it is observed that similar data occurs in the tactile modality, it is apparent that this putative mechanism must be a property of production modalities as a whole and not just the verbal (speech) mode. For instance in unpublished work of the author a patient was observed who had severe alexia and agraphia with little impairment of the CLS and verbal-auditory modalities. In conversation it was only slightly apparent that he had suffered brain damage (which, incidentally, was in the general parietal region) but by contrast he could not read or write spontaneously. After much prompting and encouragement the patient was induced to attempt to write and it developed that in fact he could write his name, address and hometown birthplace, all without paying attention to the pencil, his hand or the writing task. As soon as he concentrated on the task he failed to be able to perform. Since he could not write to dictation, nor copy, the only explanation of the behavior is that there are ictal automatisms in the tactile language modality, too. Since such utterances are generally highly over-learned (practised) as well as emotionally significant, it is quite probable that the sub-cortical modality-specific parts of the production systems can 'tap' a store of integral motor commands under conditions of stress or in other emotional contexts. There is no reason to presume a locus for these motor programs and there is no evidence suggesting one, either.

The tactile system is, analogous to the visual system, considered here to be a secondary level production system that relies on general somatic efferent patterns of input which it converts to written language: it is schematically shown in Fig.10. The relevant hand and arm muscles are innervated by the median, ulnar and radial nerves which are spinal nerves emanating from the brachial plexus. The motor nuclei of these nerves are controlled *via* the corticospinal fiber tracts which are generally identified as the Pyramidal Motor System. The *pyramidal* tracts are, incidentally, phylogenetically a late development in man and the primates; the so-called extra-*pyramidal* system is the older one. The *pyramidal* tracts descend from the motor cortex anterior to the *Fissure of Rolando* from a

Fig.10 Anatomical schematic of the tactile modality.

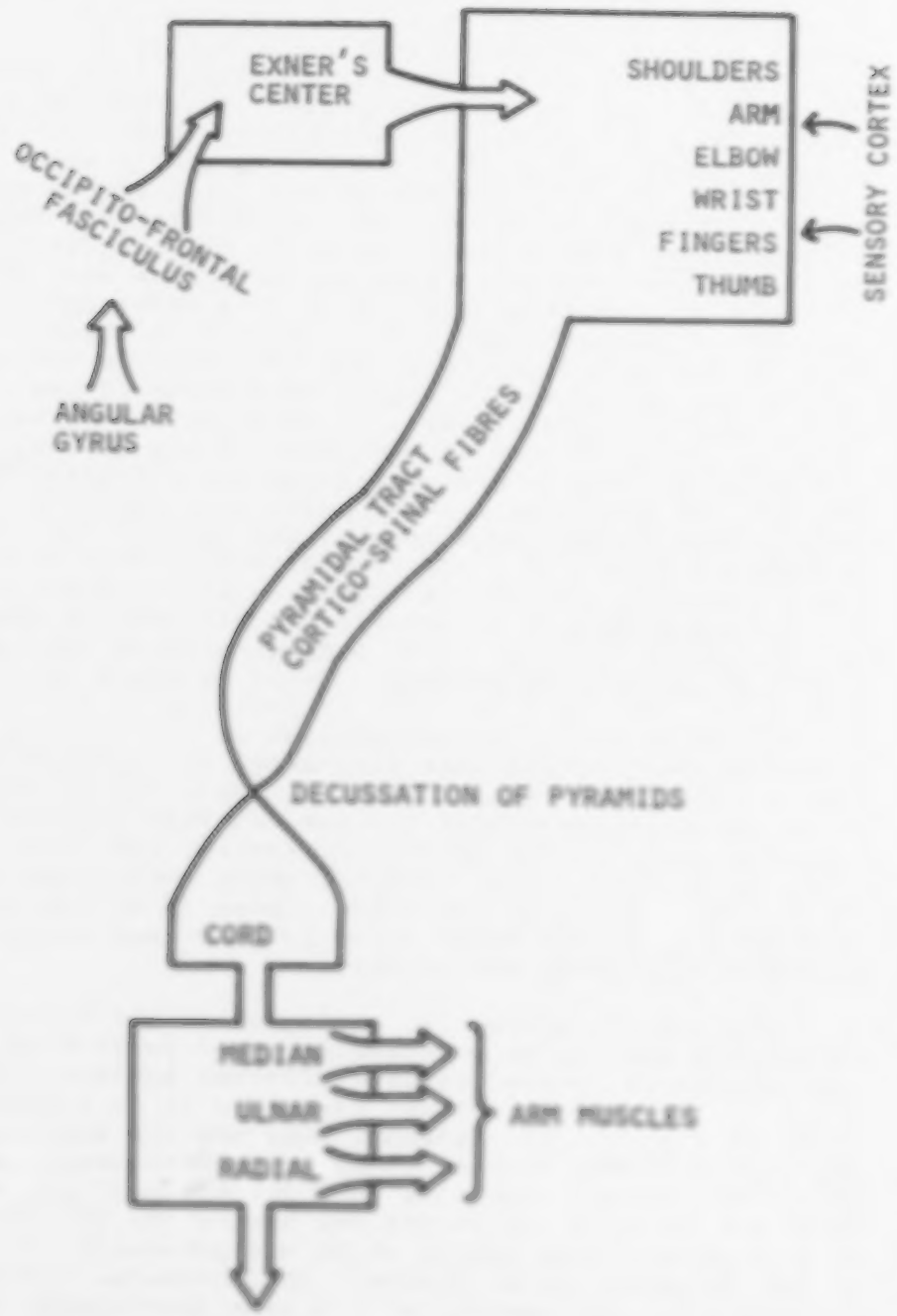
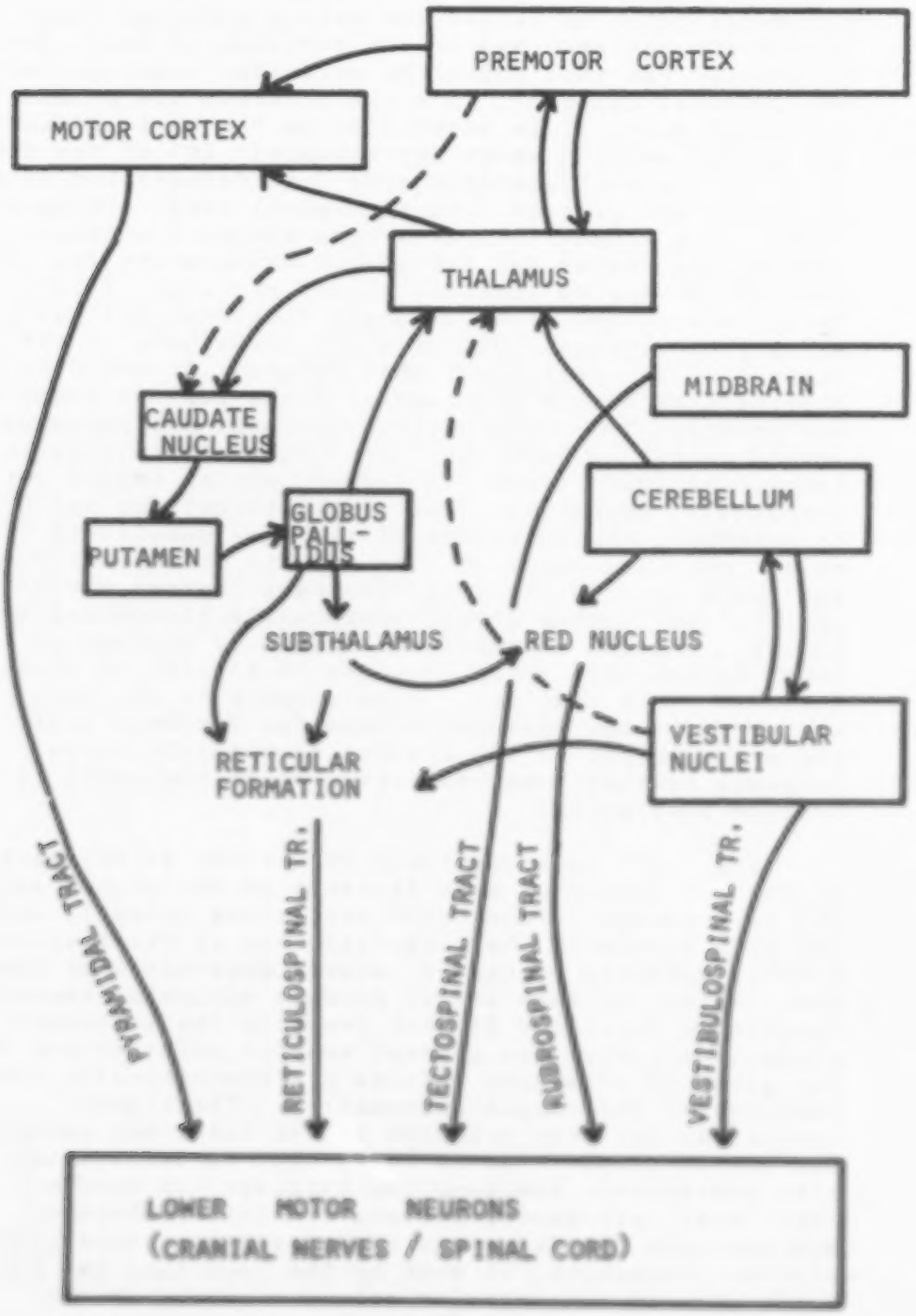


Fig.11 Anatomical schematic of the extra-pyramidal systems



point just above the area designated as vocal-tract (motor) in Fig.4. The tract first finds its way through the fiber tracts of the corpus callosum (at this point the pyramidal tract is called the corona radiata), then around the thalamus and corpus striatum or basal ganglia structures (at this point the pyramidal tract is called the internal capsule), into the midbrain and pontine areas (at which point it is identified as the basis pedunculi) and to the medulla where approximately 80% of the fibers cross to the contralateral side (the decussation of the pyramids) and proceed into the spinal cord. These are rather long fibers - there are no synaptic connections between the cortex and the spinal motoneurons for this part of the system (usually called the alpha fibers). There is a second group of alpha fibers as well as a gamma motor fiber system, both of which contribute to the control of the peripheral musculature. It would be difficult to adequately summarize the various motor systems and putative functions which supplement the pyramidal system except to note that they first of all involve the extra-pyramidal tracts and second, act to smooth out, coordinate, integrate, control the target for and hold in position, the multitude of skeletal muscles in the body. For discussion of some aspects of these systems and their role in the local feedback control circuits for speech, see Tatham [1969], Hardcastle [1970] and Reid [1970]. A gross schematic of the basic anatomy at the supra-spinal cord level is shown in Fig.11; it is not intended to be complete. With respect to the possible use of the gamma efferent system for feedback control of the motor output of the cranial nerves (the verbal language system) some comments will be presented in section 5(d) below.

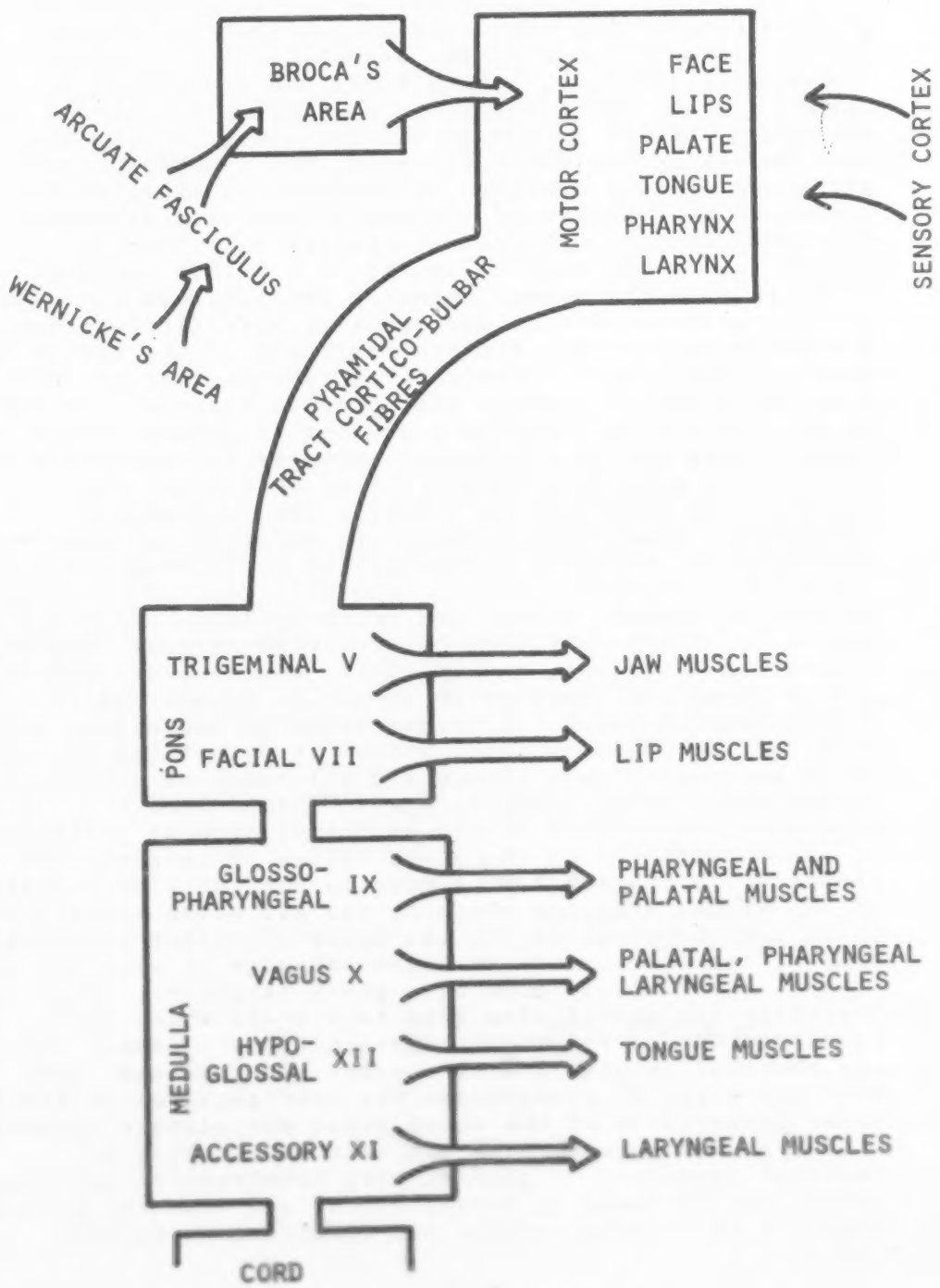
The lower (sub-cortical) structures of the tactile system are not of as much interest as the higher structures for two reasons. First they are system-specific and do not play a role in the linguistic use of the tactile-graphic modality as can be readily determined by the fact that lesions in them do not produce agraphia; second, linguistic functions at this level in the secondary production system are general somatic efferent and thus any group of voluntary muscles are theoretically capable carriers of the output information. The higher structures are very relevant to the model but unfortunately are not so simple. It is known that handedness correlates with hemispheric dominance for language but *need not* - in fact, most left-handed persons are left-hemisphere dominant for the CLS. Since the pyramid fibers cross the midline (decussate) it must be the case that the hand motor area in the anterior rolandic cortex in the

contralateral hemisphere controls the preferred hand. For left-handed persons this will generally imply that the right hemisphere motor cortex controls the hand while for the same person the left hemisphere motor association cortex controls the linguistic use of the hand; therefore, the signals to the tactile system must cross the corpus callosum for such persons, which is a rather inefficient manner of achieving things though certainly possible. Nevertheless, since the tactile language modality makes use of a general somatic efferent signal, it may make very little difference which muscle groups activate it or in which hemisphere they are cortically represented. There is good evidence in addition that the foot of the middle frontal gyrus, identified as Exner's Center in Fig.4, as well as the occipito-frontal fasciculus and some part of the Angular Gyrus, all play a role in the tactile system; for lesions in any of these areas can produce agraphic symptoms that are virtual analogs of apractic symptoms caused by lesions in Broca's Area or the arcuate fasciculus. Since Exner's Center is motor association cortex, as is Broca's Area (which incidentally, interconnects with it *via* short cortical fibers), and since the *primary* motor cortex anterior to the Fissure of Rolando does not connect *via* the corpus callosum with the other hemisphere, whereas fibers from Exner's and Broca's areas do so connect, it may be possible to fit these complex systems into a plausible framework. From Exner's Center which is the main staging area for sending signals to the tactile language modality, information flow may follow several possible routes to the appropriate production sub-systems: the corpus callosum to the motor association cortex in the opposite hemisphere to the primary motor cortex and then down the pyramidal system, directly to the primary motor cortex within the same hemisphere and then on down the pyramidal system, or possibly directly down the pyramidal system from Exner's Center itself, either side. At the moment there is an insufficient amount of evidence available to adequately distinguish these possibilities.

In any event we might note the following general characteristics: the primary motor cortex (anterior rolandic cortex) is labeled in terms of specific parts of the body and muscle groups, which in effect means that if this area does figure into the production systems (either tactile or verbal) then there are labeled transmission lines from the cortex to the appropriate musculature; motor association cortex such as Exner's Center or Broca's Area is not so labeled. There appears to be the possibility then of a three-stage model for the tactile system, which in two particular respects will be quite similar to the verbal language modality to be discussed next, as follows:

the first stage involves the tracking of linguistically significant and phonologically specified graphic patterns by mechanisms in Exner's Center, where by tracking is meant specifying the appropriate general somatic efferent signals which will ultimately direct peripheral muscles to achieve graphic shape targets. The time-base characteristics of the tactile tracking mechanism is obvious not as crucial as that for the verbal tracking mechanisms; nevertheless, it is obvious that the rate of display in the CLS buffer and the rate of tracking by the tactile buffer may not be the same and in fact may be voluntarily varied. The second stage would be the organization of the general somatic efferent signals into commands appropriate for the muscles of the intended system to be used; by this it is presumed that the graphic pattern is recoded three times: linguistically, general motor efferent, and as specific muscle commands. The verbal language modality probably does not have the second - general somatic efferent - recoding stage since signals in this system are system-bound as has been repeatedly noted. The third stage in the tactile model is, of course, transmitting the signal through the correct fiber tracts. Up to this final stage it is presumed that there is linguistic control of the somatic efferent system and that lesions in structures responsible for these stages will lead to agraphia. If the tracking system is not functioning properly one would expect the output buffer to produce on occasion some graphic shapes that are appropriate to the language but erroneous in the specifically intended context. This does happen and in fact parallels apraxia as noted before. Finally, from the point of view of the model, it is difficul to find evidence for the second stage - the organization of the signal into appropriate general somatic efferent signals since the concomitant of impairment here would actually lead to no production at all. By contrast there is striking evidence for the third recoding state in the verbal system as is seen in the dysarthrias. One might wish to take as evidence for the second stage such facts as whether the writing is evenly spaced, more or less level to the page, and whether the letters are in fact letters or meaningless scribbles, all within certain tolerances of course; some patients do write with minute nearly-illegible scrawls that fall off sharply to the right or the left and it is unlikely that they could produce any writing of a better quality with, for example, the other hand, or by grasping a pencil in the teeth, etc. An adequate account of the modality-specific aspects of the tactile system will probably require, among other things, a plausible theory of writing, replete with 'dialect variation' and all.

Fig.12 Anatomical schematic of the verbal modality.



5(d). The Verbal Language Modality

The verbal language modality has been studied in greater detail than any other aspect of the language system simply because of the accessibility of data of a linguistic, acoustic, articulatory and psychological nature. Since the literature in linguistics, phonetics, neurology, speech pathology and psychology is replete with models and evidence for them, the remarks to be made here will be confined to emphasis on the somewhat different perspective of the neurolinguistic framework developed so far. The verbal production system is schematically represented in Fig.12. Within the verbal language modality we may identify the clearest differences between system-specific and modality-specific functions. The evidence for this distinction rests on the easily observed behavioral correlates of lesions that produce dysarthria and of lesions that produce apraxia. In the former the system loses to a greater or lesser extent its control over the speech musculature *per se*, resulting in deficits ranging from total loss of speech to, for example, the inability to modulate the fundamental frequency. Dysarthric affects can separate out numerous physiological phonetic parameters of vocal-tract control - nasality, whisper/voicing, adequately forceful closure of lips or tongue, intra- and inter-syllable durations, degree of stress, phrasing between words (pause length), intonational contour, or, in short, nearly every muscle, muscle group and combination of muscle groups can be selectively isolated. A proper study in neurolinguistic terms of the phonetic implications of dysarthria has yet to be undertaken even though the relevance of such a study is obvious. Among other things it should lead to hypotheses which have strong empirical support pertaining to which processes in the sound system of language are phonetic (in the sense of common to the functional anatomy of the verbal language modality for all human beings) and which are phonological (in the sense of either universally true of the CLS aspects of human language or else and more likely, particularly true of a given language). Such an investigation should also lead to a model which uses features that correlate with articulatory commands and the neuronal impulse trains responsible for them; such features might be grounded on the configuration of cranial nerve innervation of the vocal tract musculature together with hypotheses concerning the nature of coding in neuronal impulses. A good general treatment of the cranial nerves can be found in Brodal [1959] and some the possible codes in the nervous system are discussed in Bullock [1967].

As implied earlier, lesions which produce dysarthria rarely if ever disrupt cortical areas - they are essentially sub-cortical. Furthermore, they are usually lesions of the basal ganglia and other upper motor neuron nuclei (pseudo-bulbar palsy) or they are lesions of the brain stem and other lower motor neuron nuclei (bulbar palsy), or they involve systems in the cerebellar circuits (ataxias). Therefore they provide good evidence that the distinction between modality-specific and system-specific functions are anatomically as well as behaviorally well-founded.

Damage to the cortical areas of the verbal language modality, in particular Broca's Area and the Arcuate Fasciculus, produces apraxia of speech which is the deficit that isolates the linguistic use of the verbal modality from the system itself. One of the most salient features of such a deficit is the rigid adherence to the laws of phonological organization of the particular language regardless of the severity of the dysfunction. This was carefully observed by Lecours and Lhermitte [1969] and also by Green [1969a]; Green notes:

> With hardly any exception the patient's paraphasias and jargon episodes consist of English phonemes. Occasionally consonants and vowels such as /f/, /s/ and /a/ are overly long; there are a few instances of unrounded back vowels and rounded front vowels, and irregular consonant clusters... But in the main the paraphasias are ruled by the phonological structures of English. All the English phonemes occur ... at very much the same frequencies that one finds in American English... Neologisms contain twice as many consonants as vowels, just as English words do, and they average about six or seven phonemes long. (106)

The full linguistic relevance of apraxia may be appreciated from a brief summary of the kinds of dysfunction that have been observed: systematic shifts from one consonant type to another as, e.g., changing affricates, nasals and continuants into stops (Blumstein [1968]), confusions with complex consonant clusters leading to either extra syllables being introduced or substitutions or simplifications of target clusers (Darley [1968]), misplacing of stress reflecting in many cases an erroneous analysis of the grammatical form class, errors in vowel reduction and consonant/vowel alternations associated with English derivational affixes, substitutions of words based upon a quantifiable degree of phonetic similarity, or changing the specific derivational affix in order to use

a word of a grammatical class that is more easily manipulated as, e.g. in changing verbs into derived nominals because nominals are easier to process.

At least some of these errors committed by apractics indicate malfunctioning in a mechanism previously identified as a tracking or guessing strategy in the other three language modalities. In the case of the verbal production system we might assume that it tracks at some point the final buffer of the CLS (in linguistic terms, the level of systematic phonetics) providing an articulatorily well-motivated set of efferent commands to the verbal language modality as a match for a linguistically well-motivated set of signals from the CLS. If the parameters employed at either end failed to be fully specified, the figures on selection and copying errors would naturally soar well beyond the level normal for relaxed conversational speech. If the tracker could search the CLS buffer both forwards and backwards as necessary and within the allowable time limit, it seems reasonable that much of the data on reversals (spoonerisms) for example could be seen as either an anticipated change based upon a scanning jump or a residue that was re-scanned unnecessarily.

The model adopted here considers both the Arcuate Fasciculus and Broca's Area to be intimately involved in the phonological component of the CLS. Following a suggestion of Blumstein [1968], it is likely that the Arcuate Fasciculus is more involved in transferring abstract phonological information from the CLS to the tracking mechanism than it is in specifying the correct motor programs with which such information will be associated. Broca's Area - the inferior frontal gyrus - would then be the potential candidate for the locus of the tracking mechanism. This view fits, by analogy, the postulate that all guessing and tracking strategies are located in cortical staging areas where the four peripheral modalities interface with the CLS, as originally suggested by the placement of the circles in Fig.2.

6. Conclusion

The preceding sketch has attempted to propose a sensible model of language incorporating what appears to underlie our ability to speak and comprehend; correlated with this proposal has been a series of speculations on the functional neuroanatomy of each putative component. One of the more important conclusions to be drawn from the model as formulated is the nature of the relationship between central and peripheral language systems and a brief review of this might usefully put it into proper perspective. The evidence alluded to in the preceding sections seems to indicate that each of the four peripheral language modalities may be considered both in terms of their respective functional anatomy (system-specific functions) and their use as input and output systems for the language mechanism (modality-specific functions). A consideration of the structures and their operation of the central language system indicates that each of the four peripheral modalities interfaces with the CLS in a specific manner and in a specific place in the central nervous system. It would not be too unrealistic to assume that what have here been identified as tracking and guessing mechanisms which in a sense convert CLS linguistic representations into the modality-specific representations required by input/output functions, is in some way a reflection of one of the contributions made by the cerebral cortex to human language. To put this in another perspective, it would be interesting indeed if it turned out that cortical areas in man's brain which are homologous to cortical areas in the brains of other primates on anatomical criteria, in fact turned out not to be homologous because of physiologically different modes of functioning; a possible mode, so to speak, would be the neurological

substrate of what has been called the tracking/ guessing strategies.

Within the sphere of the CLS itself, the model proposed does not distinguish modality-specific functions. In the literature of apasiology it has often been remarked that there is no deficit which is confined solely to one modality; while a patient may exhibit predominantly receptive disorders, a careful examination will usually reveal an expressive component. Such observations fit the model which is suggested. The CLS, even though divided into several cortical fields, and even though divided into a phonological component and a remaining syntactic/semantic component on anatomical grounds, is nevertheless a cohesive mechanism in the psychologinguistic sense. The units and rules of the grammar underlie the uses of the grammar in all modalities and therefore, if the CLS is damaged, it is to be expected that the deficit will be manifest in all modalities. On occasions this can take rather striking forms, e.g., an impairment in reading and writing personal pronouns, frequent error in the use of personal pronouns in spontaneous speech, and an imperfect awareness of the specific features of the personal pronouns in comprehension; when explored in full detail even a slight deficit if it is a CLS deficit can be shown manifested in all four peripheral modalities. Taken together it is hoped that the above ideas form an adequate basis for neurolinguistic investigations. The many gaps in the model and the weaknesses of may of the ideas do not need to be reiterated; the reliance on previous work, much of which is cited in the references, is apparent in most of the hypotheses which have a fair degree of empirical support. The overall synthesis may have a number of useful consequences in addition to hopefully advancing the understanding of language. Most studies of aphasia that the author happens to be familiar with may be fit into the proposed framework without violent distortion.

At least one minor benefit of reconsidering the aphasiology literature in these terms is that it would obviate the need for a plethora of terms and definitions which at the moment appear to be impeding our understanding of the brain rather than advancing it. The possibility of incorporating evidence from aphasia into the domain of linguistic theory is implicit; the neurolinguistic framework clearly indicates how such data may be considered and a number of the cited references deal specifically with such data in terms of linguistic hypothese. A strong claim for the model is that it may be able to decide some issues in linguistic theory on

the basis of evidence from a wide domain of investigation. As such it may be regarded as enriching the explanatory power, in the ordinary sense of the word, of linguistic hypotheses. Even if this claim is not fully realized, the neurolinguistic model is a step towards an adequate theory of performance, which obviously must be grounded in the neurological sciences if it is to be at all successful. With the more complete specifications and hypothesizing that are to be expected with further research along these lines, it may very well develop that the need for highly abstract theories in linguistics will no longer exist; instead one might expect functional equivalencies in the domain of the neurological sciences to provide the behavioral corroboration that now seems to be lacking.

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